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THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY

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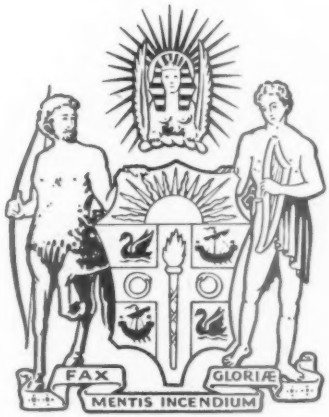

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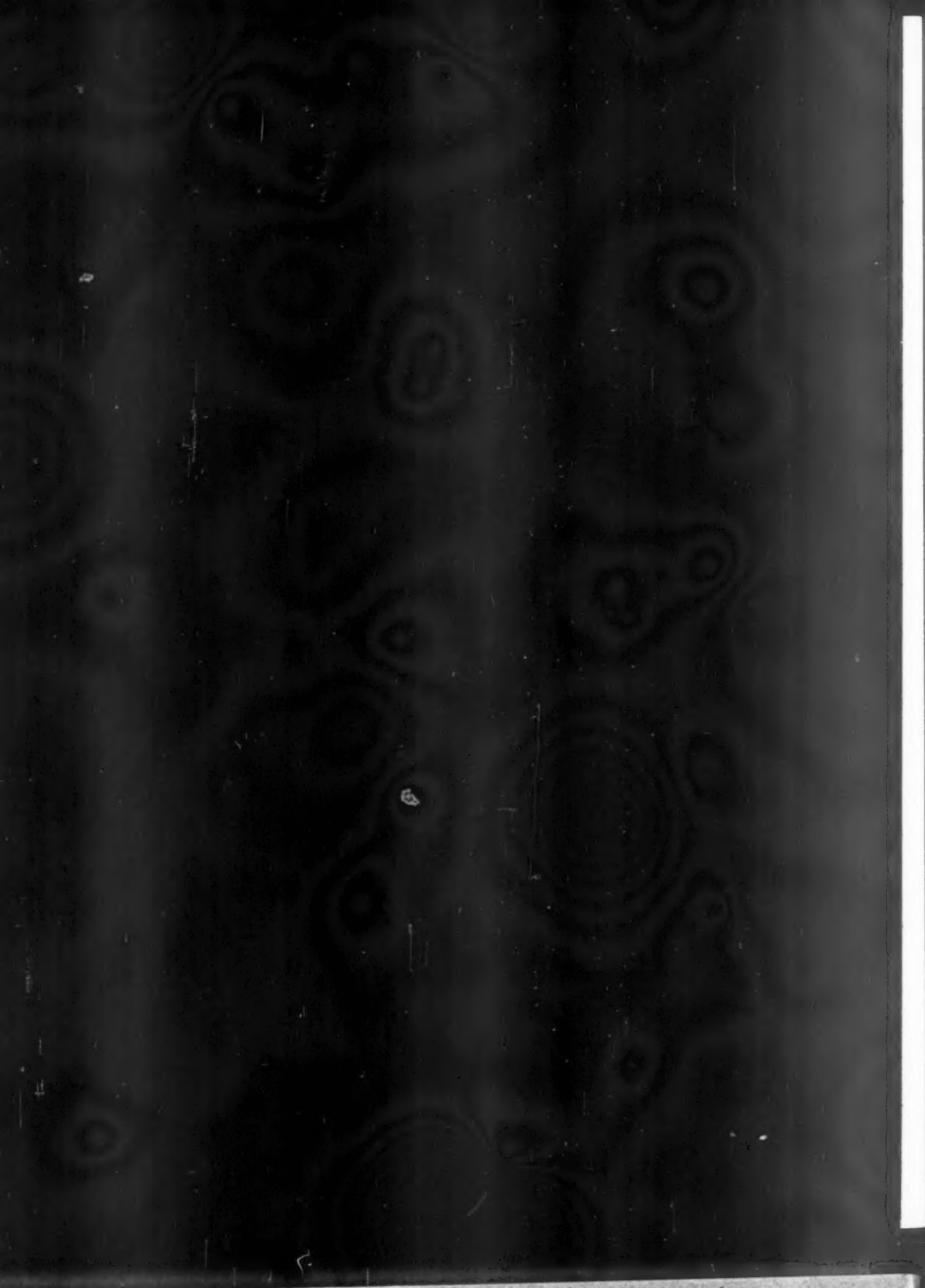

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The Australian and New Zealand
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Vol. XXV — No. 1 AUGUST, 1955

A STUDY OF PIGMENTED SKIN LESIONS*

By J. D. Hicks, B. K. Rank and A. R. Wakefield

The Royal Melbourne Hospital

THERE is every indication that the whole medical profession is prone to bewilderment and fear when advising patients about common pigmented skin lesions. This is a highly responsible task in which we have all often been unsure in observation, interpretation and advice. Many escape by a policy completely non-committal, referring all these cases to a consultant. He in turn has been liable to pass all responsibility on to a pathologist with blind acceptance of his report as the basis for prognosis or action. Little have we realized the relative and static nature of many of the pathologist's criteria which so often render his report of doubtful value. Other practitioners, with equal lack of confidence, regard every pigmented lesion they see with suspicion—they say it is better to be sure than sorry—and all comers are subjected to wide excision operations. A spreading fear complex among the lay public is the corollary of this attitude and its end is the ridiculous.

In a short span of consultant practice we have been struck by the number of patients with pigmented skin lesions referred by colleagues of all branches. The high proportion among these of doctors themselves, their wives, families and immediate associates is testimony of the common alarmist attitude which exists. If surgeons are not to be mere technical automatons, or if on the contrary, and perhaps despite previous advice, they are to re-assure a patient that a given lesion implies no special risk of malignancy, then it seems that a much better general understanding of the clinical nature and behaviour of these lesions is necessary. In an organized endeavour to this end the writers have taken advantage of three circumstances:—

First, a large number of these cases (some 321 cases†) which have come our way in the past ten years have been subjected to close personal study, observation and record. It is perhaps noteworthy that the long list of writers who have contributed to this subject includes few, if any, who have had continued personal clinical observation of the patients with the lesions they have studied histologically. Most have access to one particular facet of this problem and so it is that theories, nomenclature and classifications have become hopelessly entangled.

Secondly, we have taken advantage of the micro-projector —a present-day highlight in bringing surgeon and pathologist into close working harmony. Thus we have been able to comb through our specimens separately and together, with and without clinical facts of each case. The histological features of 220 lesions have been detailed and checked by the one pathologist (J.D.H.), first without reference to clinical facts, and again reviewed in relation to the clinical story and known behaviour of each case treated personally by one and often well known to both surgeons.

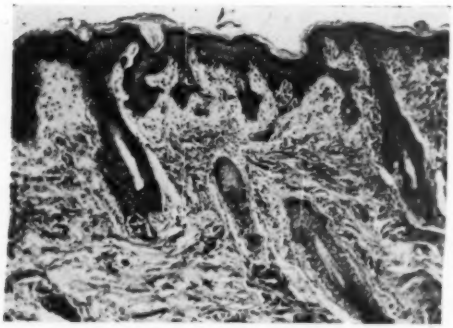
*Presented in part at the Annual Meeting of the College of Surgeons in Melbourne, August, 1954.

†The main basis of this paper however is the study of only 220 of these, as only in that number was the material available for proper histological study.

Thirdly, we have been stimulated by the published work of the American pathologists, Allen and Spitz (1953). This first attracted interest because some of their deductions as pathologists from histological study were surprisingly close to what many clinicians have felt for a long time about some of these lesions, though perhaps in rather a vague way. In other words, their histological criteria of activity and malignancy indicated a closer correspondence with known clinical behaviour. We were keen to see if their observations applied to our cases.

(a)

(b)



(c)

FIG. I. (a) Junctional naevus with typical junctional cells and groups of cells situated along the basal layer of the epidermis. (x 86)
(b) Intradermal naevus where the cells are situated wholly in the dermis and are separated from the basal layer of the epidermis by a thin layer of connective tissue. (x 86)
(c) Compound naevus where the cells are seen predominantly in the dermis but junctional cells and groups of cells are still present. (x 86)

NOMENCLATURE AND BASIC HISTOLOGY

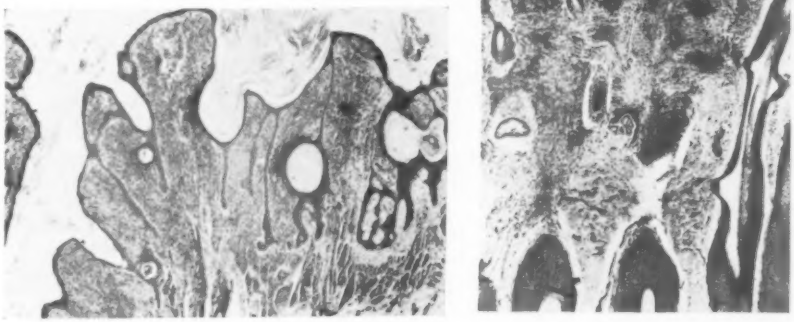
Classification can become a disease in itself —instead of clarifying it may confuse. Of this there is no better example than in relation to pigmented skin lesions, the nomenclature of which has been built on the crossfire of classifications elaborated by dermatologist, surgeon and morbid anatomist.

We have found it impracticable to free ourselves from terms which have been associated in the past with more than one meaning so that it is necessary to define what we mean by the terms we use.

There are fundamental objections to the use of the terms "mole," "naevus," and "melanoma" for innocent pigmented skin lesions. "Mole" implies a three-dimensional lesion, "naevus" refers to any skin blemish, usually congenital, and "melanoma" denotes a neoplasm. We select the following terminology as the most satisfactory. The large groups of innocent melanin-pigmented skin lesions, whether congenital or acquired, we call pigmented naevi. We retain the word naevus only because it is so established and ingrained that it is deemed unwise to suggest any alteration. Furthermore, in so doing, we purposefully avoid using the term melanoma for pigmented lesions which are not neoplasms. By melanoma we mean malignant melanoma.

To appreciate the basis of this paper, some account is necessary of the various types of pigmented naevi and the stages of their formation and maturation. The conception of a life history of the pigmented naevus and various aberrations thereof is fundamental.

In most pigmented skin lesions there are collections of small cells, round or oval in shape with small dark nuclei which resemble but differ slightly from the basal cells of the epidermis. These "naevus cells," as they are generally called, or melanoblasts (the term we prefer) may occur singly but are more frequently in clusters. These cells, readily demonstrable by ordinary staining methods in pigmented naevi, cannot be so demonstrated in normal skin, in the skin of normally pigmented areas, or in the skin of dark-skinned races. Naevus melanoblasts in contact with the epidermis are termed, from their position, junctional. In this situation they are seen as swollen vacuolated cells sometimes containing fine pigment granules. Those which are in the dermis and separated by a layer of connective tissue from the epidermis, are called intradermal. Where only junctional cells are present, the lesion is termed a junctional naevus. Where only intradermal cells are present it is termed an intradermal naevus, and where there are both junctional and intradermal cells the lesion is termed a compound naevus (Fig. I).



(a) (b)

FIG. II. (a) Intradermal naevus with well-marked papillary and cornified surface. (x 50)
(b) Intradermal naevus with numerous hair follicles and sebaceous glands. (x 36)

Pigmented naevi have a definite life history. Thirty years ago J. K. Dawson (1925) showed clearly how the melanoblasts first appear amongst the basal cells of the epidermis, generally in groups, and how they migrate into the dermis to form the typical cell masses seen in these lesions. Thus, in the earliest stages they are junctional in type and as some junctional cells separate from the epidermis, they become compound. While junctional cells are still present the naevus is immature. When junctional cells are no longer found it is mature and entirely intradermal. Atrophy and fibrosis with separation of cells can be seen in the deeper portions of most mature pigmented naevi. In some naevi the structures resembling nerve bundles and specialized nerve endings as described by Masson (1951) can be clearly seen. Many pigmented naevi show other abnormalities than collections of melanoblasts. The surface is often papillary or mamillary. Cornification may be excessive. Sebaceous glands and hair follicles may be numerous (Fig. II).

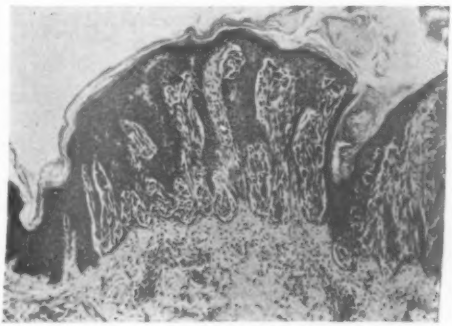
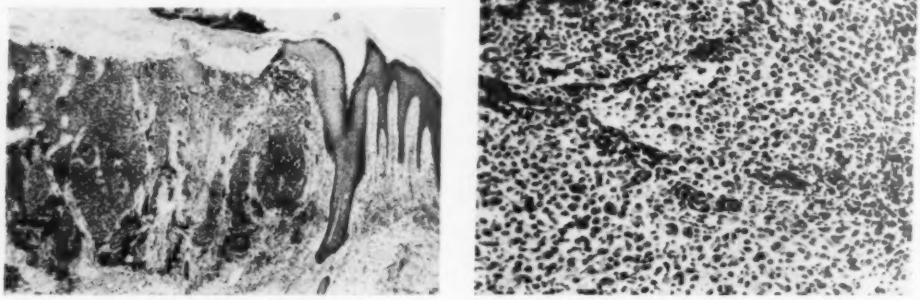


FIG. III. Activated junctional naevus. Note the proliferation of spindle cells in the junctional cell groups which are beginning to disrupt the epidermis and are bulging into the dermal papillae and down into the dermis. (x 86)

Aberrations from the normal maturation process are many. It must be realized that the life history of a naevus may commence at any age. By no means do they all begin in pre-natal or early life, and even those that do are not all mature by adult life. Furthermore, many junctional naevi never proceed to maturity at all, but either remain inert throughout life or undergo an active cellular proliferation which may be the precursor to malignancy. Junctional naevi undergoing such cellular proliferation are referred to as *activated junctional naevi* (Fig. III).



(a) (b)

FIG. IV. (a) and (b). Typical examples of malignant melanomata where the diagnosis is beyond dispute. The criteria of malignancy can be seen, including wild cellular proliferation with mitotic figures, alteration of cell characteristics in size, shape and staining, and extension of cells beyond normal bounds with surface ulceration and deep extension into the dermis. (a) (x 36) (b) (x 144)

One typical and particular form of junctional naevus is a diffuse spreading lesion which first appears in adult life. It does not mature but on the other hand is prone to continued active proliferation in the basal layer, often as a precursor to subsequent malignant change. This diffuse form of junctional change we call *diffuse juctional naevus*. It corresponds to the clinical entity of melanosis.

Malignant melanoma

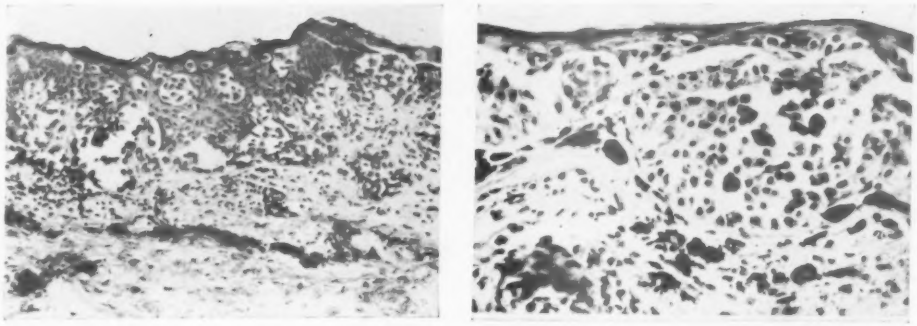
It is of singular importance to be precise about what we term "malignant." It is just this which is significantly absent from so many clinical reports about "melanomata" and which makes all figures and comparisons futile. Furthermore, we believe it is because of failure to establish working criteria of malignancy that the term "melanoma" conveys a feeling of uncertainty which pervades all thought and writing on these conditions. Whether writers agree as to these criteria is of little account as long as they are clearly stated.

The histological criteria of malignancy do not differ from those of other tumours. There are two main features which must both be present.

(1) The general cell characteristics of malignancy — proliferation of cells and alteration of cell characteristics, of size and shape and staining, with enlargement, irregularity, and mitosis of the nucleus. There may be increased cellular function, i.e., melanin formation.

(2) Extension of cells beyond normal bounds both upwards to cause disruption of the epidermis and downwards as sheets of cells into the dermis.

In most malignant melanomata these criteria are beyond dispute (Fig. IV). In some cases, however, while there may be alteration in cell characteristics suggesting malignancy it may be difficult to determine whether cells have extended beyond normal bounds. The distincion between the earliest malignant change and simple activation of a junctional naevus may be extremely difficult, and depends on the identification of malignant cells in the dermis which are clearly separated from the epidermis. Where invasion is confined to a narrow band immediately below the epidermis the term superficial malignant melanoma is used (after Allen and Spitz) (Fig. V).



(a) (b)

FIG. V. (a) Superficial malignant melanoma. Single cells and groups of cells have extended up through the epidermis towards the surface breaking up the normal pattern. Other small groups of cells have broken away from the epidermis and can be seen lying separated from the epidermis in the superficial portion of the dermis. (x 144)

(b) Superficial malignant melanoma under higher magnification to show the cell characteristics which suggest malignancy. (x 288)

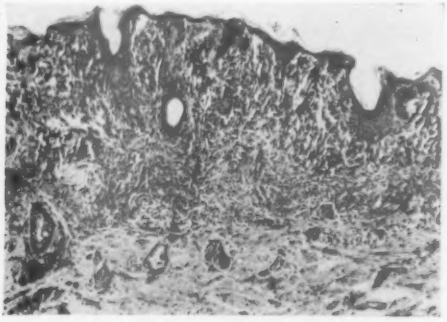
Juvenile melanoma

This has been described as a separate entity by Spitz, and is said to be characterized by an accumulation of spindle cells or loosely arranged irregular melanoblasts in the epidermis and in the dermal papillae.

The general appearance is of rather wild proliferation, and suggests a diagnosis of malignant melanoma (Fig. VI). When one learns the age of the patient, and realizes that the prognosis of such a lesion is almost uniformly excellent, one is puzzled as to the relationship between these and histologically similar lesions in adults. We have been quite unable to distinguish these lesions as separate entities on histological grounds, as Allen and Spitz claim to do. Their behaviour and clinical significance will be considered later.

The "Blue Naevus"

One uncommon variant of the pigmented naevus which has been called the "blue naevus" consists of spindle cells which are

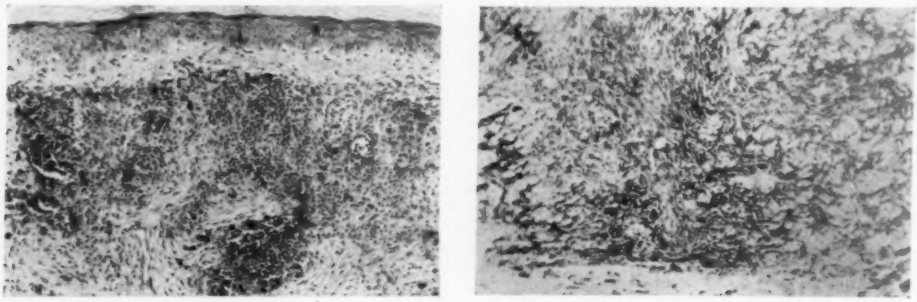


(a) (b)

FIG. VI. (a) Juvenile melanoma in a child of 3½ years.

(b) The same lesion examined histologically shows wild proliferation of spindle cells which is largely confined to the epidermis and dermal papillae. (x 86)

spread through the collagen of the dermis and even into the subcutaneous tissue. They may contain a large amount of melanin pigment, which, together with its depth in the dermis, is responsible for the blue-black appearance. Few cells recognizable as melanoblasts may be present, but from a study of several of these spindle celled lesions we think many of the "blue naevi" may have developed from ordinary intradermal naevi (Fig. VII).



(a) (b)

FIG. VII. Blue naevus, which resembles the common intradermal naevus in its superficial portion (a), but extends more deeply into the dermis and subdermal tissue where the cells are spindle shaped and different in character (b). (a) (x 144) (b) (x 86)

Thus, in summary, it is seen that benign pigmented naevi of junctional, intradermal or compound type present no difficulties in histological diagnosis. Neither do the unusual blue naevus and the condition of diffuse junctional naevus. We are unable to distinguish the condition of juvenile melanoma on histological grounds alone. The fact that similar histological conditions behave differently according to their relationship to puberty is of great clinical significance, but should not colour the outlook of a pathologist in describing what he sees. The greatest difficulty is encountered in differentiating an activated junctional naevus from a superficial malignant melanoma. In making this distinction we have relied entirely on the criteria of cell characteristics and distribution, but there is obviously no recognizable point in the transition from activation to malignancy. It may be extremely difficult to determine when cells have broken away and invaded the dermis. We have detailed our criteria of malignancy but agree that with experienced teamwork of surgeon and pathologist these might well be modified.

CLINICAL REVIEW OF PIGMENTED SKIN LESIONS

In practice, pigmented skin lesions present as certain well-defined clinical problems.

Group 1

In the first group are all those patients from babyhood to early adult life who themselves or whose parents are concerned about *disfigurement* from lesions that have existed since birth. These are generally large and vary tremendously in the degree or density of both pigment and hair. They range from extensive disfigurements covering wide trunk and limb areas through more typical patches on face and forearms to those common small raised hairy pigmented moles, the typical oval or pear-shaped lesions often seen on the face (Fig. VIII). The larger the lesion, the earlier these patients present, and concern about small lesions generally only arises when they occur on the face, and often later in life.

It is of little significance whether these are first obvious at birth or in early childhood, for all are prone to change during the growth period in regard to the depth of pigmentation, the amount of hair growth and the nature of the surface, which often gets more papillary and keratotic. Some may develop discreet areas of denser pigmentation quite different in character from the main lesion to make queer looking lesions (Fig. IX). However,

they do not change in surface extent except in relation to general body growth, i.e. the change is a maturing process and not a formative process. Histologically all these hairy pigmented lesions show a similar basic pattern — they conform to the structure of compound naevi, i.e., there is some junctional component. The younger the patients the more junctional proliferation is apparent (Fig. X), and this variation in pattern over the different age groups is significant.

(a)

(b)

(c)

(d)

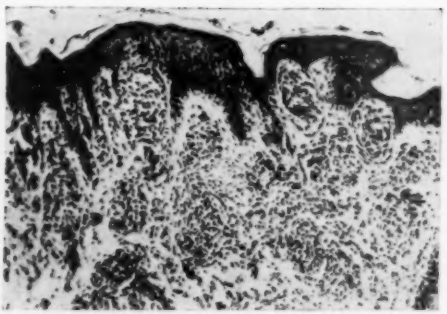
FIG. VIII. (a), (b), (c) and (d) are typical examples of congenital pigmented naevi.

FIG. IX. A curious atypical congenital lesion.

Up to the age of ten, we found very few cases in which these naevi were of mature intradermal type, and in many of them junctional activity was a marked feature. It is reasonable to assume that the long standing lesions, generally formed before birth, undergo a progressive development in early life towards the mature intradermal type of lesion, where junctional activity ceases and the naevus cell masses settle deeper into the dermis.

(a)

(c)



(b)

(d)

FIG. X. Junctional activity in congenital lesions in young patients.

(a) and (c) Macroscopic appearance.

(b) and (d) Microscopic appearance of the same lesions. (b) (x 144) (d) (x 180)

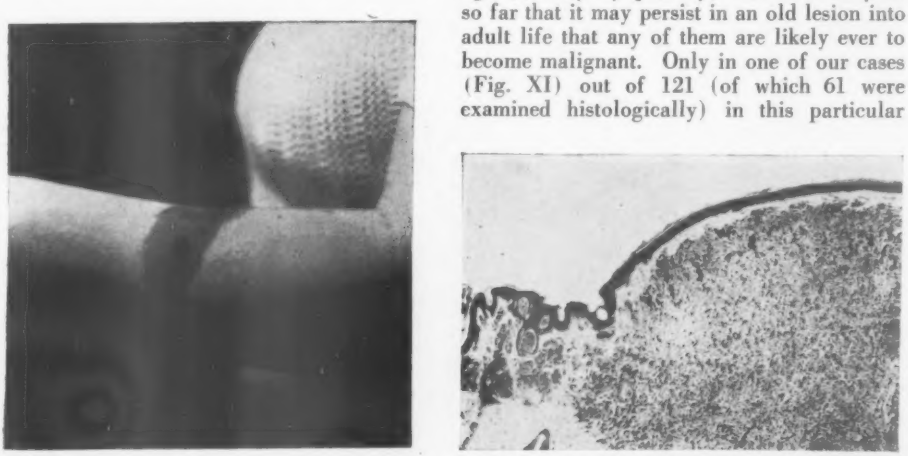
It is significant, too, that in a number of these lesions anomalies of other tissue elements are noted — nervous, epithelial or vascular — and in certain areas it is often quite difficult to make a precise diagnosis of which tissue is most affected. It seems reasonable again therefore to regard all these conditions as malformations and, if we consider for a moment the structure and functions of skin and the diverse distribution and arrangement of hair and pigment which normally occur, then it is not surprising that anomalous developments and arrangements of pigment-producing cells should occur and often be the dominant feature. Many neurofibromata are covered with pigmented skin (the pigment is generally in the basal layers), and may be confused with congenital naevi, but they make protuberant masses and subcutaneous nodules can often be felt. Whether or not some pigmented lesions associated with extensive areas of neurofibromatosis are to be called neurofibromata or pigmented naevi is largely a matter of the predominant histological picture, and does not affect the general conception of regarding all these lesions as malformations.

Table 1

The relation of histological appearance to age of lesion in a group of 61 congenital pigmented naevi

Age Group	*Junctional naevi*	*Intradermal naevi*	*Combined naevi*	*Combined naevi with junctional activity a marked feature*	*Malformations (including naevus cells)*	*Total*
Up to ten years	1	3	8	9	1	22
10–20 years	0	7	10	3	3	23
20–50 years	0	1	11	3	1	16
Total	1	11	29	15	5	61

The main question is—"do these malformations have any particular malignant potentiality?" The indication of our series is definitely to answer in general, "No," and it seems that this is because junctional proliferation and activity have ceased at an early age (usually by puberty) and it is only in so far that it may persist in an old lesion into adult life that any of them are likely ever to become malignant. Only in one of our cases (Fig. XI) out of 121 (of which 61 were examined histologically) in this particular



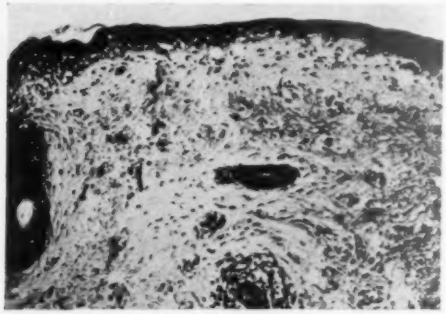
(a) (b)

FIG. XI. (a) and (b) Malignant melanoma developing in a congenital pigmented naevus where junctional activity persisted after puberty.

(a)

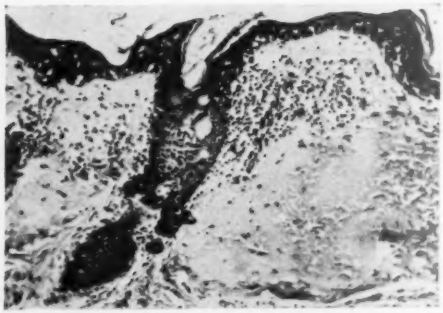
group of congenital lesions has malignancy been found—and this particular lesion did demonstrate the unusual feature of persistent junctional activity. It is the only malignant melanoma we know of arising from one of these congenital pigmented naevi or malformations, whichever we choose to call them.

In general we can state therefore that we have a group of congenital pigmented naevi which by adult life are mature and therefore



(b)

(c)



(d)

FIG. XII. (a), (b), (c) and (d) Typical examples of melanosis showing diffuse junctional activity in elderly women.

not prone to malignant change. Moreover, they show the typical clinical features of maturity, i.e., they are protuberant or nobbly, hair growing, and do not spread.

Group 2

Here again is another typical clinical group seemingly little related to the first, but presenting an opposite and contrasting clinical picture, i.e., that of diffuse spreading areas of pigmentation in adult life (Fig. XII).

This is a clear-cut clinical entity which deserves much more attention than it has received in the past. These lesions occur typically on the cheek region of females in the post-menopausal age. Whether or not these areas arise *de novo* or from pre-existing pigmented patches, they show diffuse irregular and spreading skin pigmentation without raising of the skin surface. They are usually described as starting like a freckle. The pigmentation is erratic and irregular in its outline, often fading imperceptibly into normal skin. The closer one looks at these areas the more difficult it is to define their limit, and often there are diffuse outlying patches in the same state.

The importance of lesions in this group is threefold: (i) They show in obvious form the clinical appearance which corresponds with the histological picture of a junctional naevus, and it is from their study we can best learn to discern the lesions most likely to show junctional activity, i.e., the lesions which are potentially malignant. Their appearance is quite different histologically from pigmented skin in dark races, freckles, or normally pigmented areas such as the areola of the nipple. In these latter areas the pigment is in normal basal cells and we do not find even a sparse distribution of junctional cells, i.e., there is no junctional proliferation. (ii) It would seem that most of these areas ultimately do become malignant if the patient lives long enough. They might well be regarded as "pre-malignant melanomata." Looking at many of them microscopically one can study the imperceptible transition from junctional activity through borderline and indeterminate states to a clear-cut histological diagnosis of malignancy. It would be no exaggeration to describe these areas of "melanosis" or diffuse junctional naevus in elderly people as one of the most pre-malignant conditions we know. The change is insidious. (iii) Effective removal of these lesions requires radical surface excision on account of their ill-defined and multicentric distribution. Their spread over a wide and ill-defined area, toegther with a strong tendency to recurrence at the periphery after apparently wide excision would suggest an unstable state in the area. If we add to this the sex incidence, the time of life, and the observed effect of endocrine activity both on existing pigmented naevi and normally pigmented areas, there is strong suggestion of a hormonal basis to the condition. We have seen melanosis recur in diffuse and multicentric fashion on a migrated flap in an area of excision, and also on free grafts. Without exception, all of the 20 cases diagnosed as melanosis have shown typical features of junctional activity over the whole area, and in six of them superficial malignancy is clearly apparent. If we add to this all the cases clinically diagnosed as malignant melanomata in pre-existing areas of melanosis the overall picture is highly significant.

Group 3

We now pass from conditions typically presenting at the extremes of life to the main clinical group of lesions which generally present in the middle decades—the hundreds of small pigmented spots, single or multiple, brought to notice for diverse reasons.

Table 2

Reasons for removing 129 small pigmented skin lesions

History of recent enlargement	43
Subjection to injury and bleeding	17
Repeated infection	3
Cosmetic concern	30
Cancer phobia or claim	25
Failure of some previous local treatment	4
For diagnosis	7
Total	129

It is in advising about these that most of us become confused; but if we look at them critically with the lessons of the first two groups in mind, we are in a position to see some daylight, for we can straightway sort out these small lesions into two groups of practical significance.

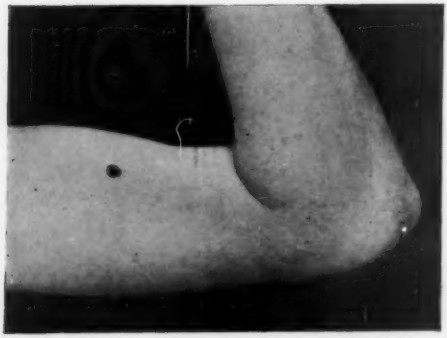
(a) The first of these are the mature lesions—the "common or garden" moles—which are like the large congenital lesions, and only differ from them in regard to size (Fig. XIII). They too are raised and generally hairy. Indeed, there seems little point in subdividing these things into congenital or acquired, nor is it possible always

to do so, as in many cases the patients have no real idea how long they have existed. Some are known to have been present since birth but there is certainly no lower limit to the size of congenital lesions. Because a lesion is not apparent to the naked eye, it does not mean it has not been present a long time before the changes associated with its gradual maturation have made it obvious.

FIG. XIII. Typical mature intradermal naevi.

The most common story we hear of these small moles is that "they have been present for years" or "as long as I can remember." These are the clinical lesions which correspond with the histological intradermal naevi. They are of no serious consequence in regard to malignant potentiality, and we must not be misled by changes often induced in them by infection, puberty or pregnancy.

(b) On the other hand, many small pigmented spots show the characteristics of immature lesions (Fig. XIV). Patients are characteristically vague about their origin. They are generally noted by chance as something like a freckle and there may be a story of change in the surface extent of pigmentation. These are small editions of the patches we call melanosis in the elderly ladies. Moreover, they too are flat, the pigmentation is superficial and there is nothing to feel with the finger tip. These we must learn to recognize as junctional naevi, and it is from these that the great majority of malignant melanomata arise.



(a)

(b)

FIG. XIV. Typical immature junctional naevi.

(c) Thirdly, if we become a little bit more discriminating, we will see, about the edges of some otherwise typical mature lesions, pigmented areas of junctional activity which, of course, have the same potentialities as the solely junctional naevus. These are compound naevi (Fig. XV).

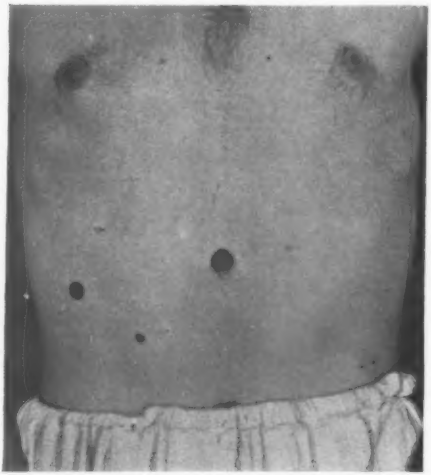


FIG. XV. Compound naevi—note the irregular pigmentation round the periphery of the main raised lesion.

Unfortunately, it is only in recent years that we have been aware of the terms "junctional activity" and "junctional naevus," so that our earlier records give little account of these clinical appearances. However, reviewing the histological features of lesions from a group of 96 cases referred to us because they were considered potentially malignant, the findings perhaps are significant.

Thus of 36 lesions which had the appearances of mature lesions (i.e., lesions which we would call now intradermal naevi on their clinical appearance), and which we did not regard with any clinical suspicion at all, but which we removed only for cosmetic reasons, for diagnosis, or for reasons more politic than surgical in the face of cancer phobia or previous alarmist advice, only in two was there any junctional activity. A few of these had single junctional cells, but not junctional proliferation. On the other hand, of all the remainder, after we again deduct those lesions which were not pigmented naevi at all (these are referred to later), i.e., those whose clinical appearance suggested activity or where there had been a history of increase in size, in approximately half (20 out of 41) junctional activity was a marked feature of the sections, and some showed the histological features of superficial malignancy.

Group 4.

Finally, we have a clinical group we diagnose as malignant melanoma because of recent evidence of cell proliferation making either a new palpable mass where none existed, or an increase in size and alteration in characteristics of an existing lump (Fig. XVI).

TABLE 3

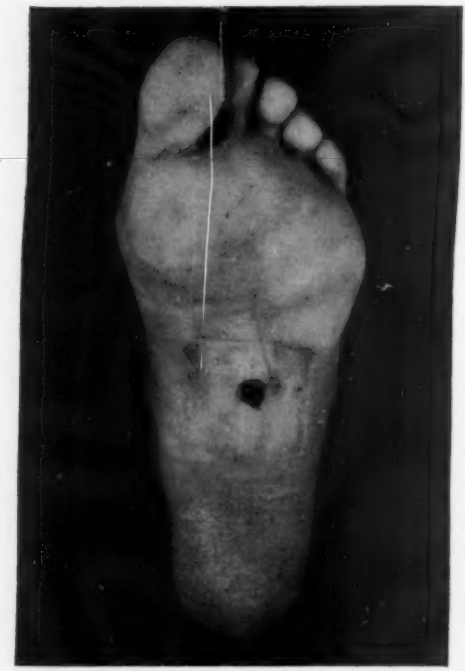
Histological features of 96 small pigmented skin lesions referred for prophylactic removal

	Malignant melanoma	*Junctional naevi*	*Junctional activity in compound naevi*	*Compound naevi*	*Intradermal naevi*	*Misdiagnosed as pigmented naevi*			
						Pigmented basal cell carcinoma	*Seborrhoeic wart*	*Pigmented papilloma*	*Angioma*
GROUP A Without suspect history or appearance	0	2	0	16	18*	2	3	0	2
					— 36				— 7
GROUP B History suspect	1	5	4	11	4	0	6	1	0
Appearance also suspect	7	0	3	0	6†	1	2	0	2
					— 41				— 12
					77				19

* Includes 4 blue naevi.

† Infection was apparent in 3 of these, and 2 were associated with pregnancy.

From amongst our series of 216 pigmented lesions of various types, we find that histologically 46 of them are obviously malignant melanomata. There was only one of these from the large group of congenital lesions; there were 7 cases from the group of small lesions we regarded with suspicion; there were eight cases amongst the areas of melanosis; and there were two atypical cases of amelanotic melanoma. The remaining 28 came from the group diagnosed clinically as malignant melanoma.



(a)

(b)

(c)

(d)

FIG. XVI. Malignant melanomata.

(a) Arising from a junctional naevus in a young woman, cf. Fig. VII (b).

(b) and (c) Arising in typical areas of melanosis in elderly people.

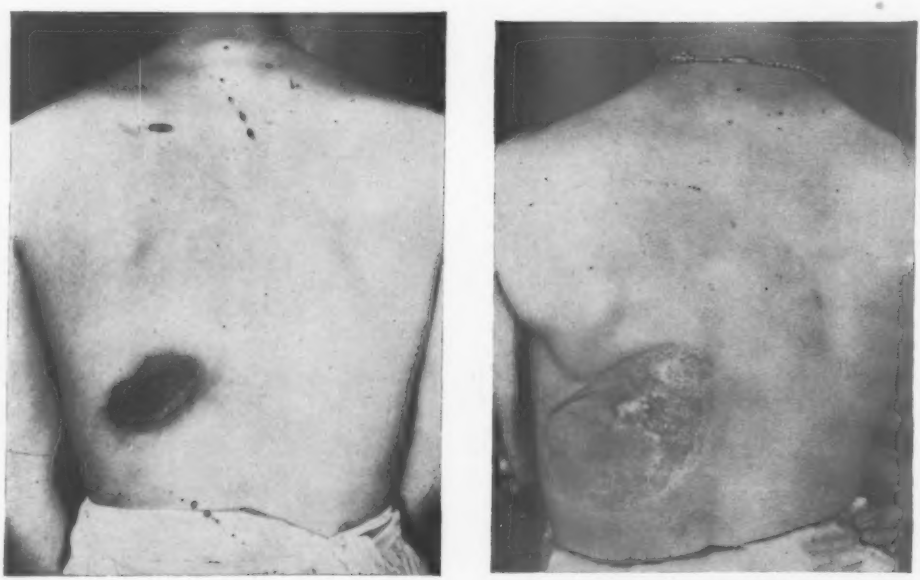
(d) Arising in a lump of many years standing, probably compound naevus.

Table 4

Histology of 42 lesions diagnosed clinically as malignant melanoma

Malignant melanoma	*Activated junctional naevi*	*Intradermal naevi*	*Other lesions*		
28	3 (1 associated with infection)	2 (1 associated with pregnancy)	9		
			Pigmented B.C.C. 3	Angioma 5	Squamous papilloma 1

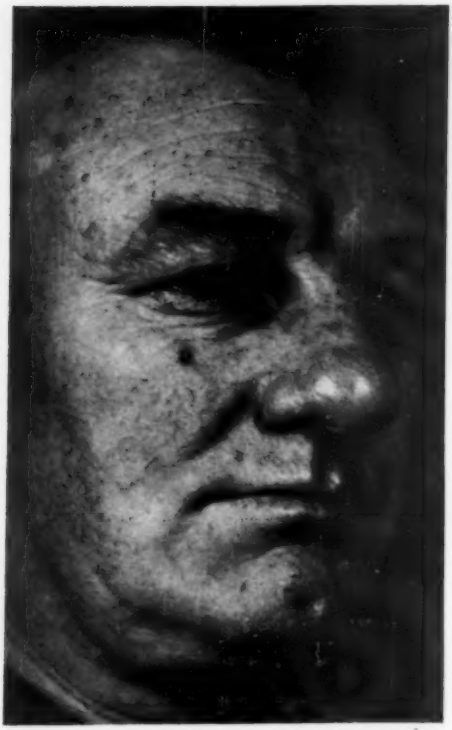
Forty-two cases were diagnosed clinically as malignant melanoma. Only in 28 cases was this histologically confirmed, i.e., in 14 cases the clinical diagnosis of malignant melanoma was wrong. Most mistakes were due to failure to appreciate the significance of benign enlargement during pregnancy, puberty or infection which, perhaps, in recent years we have been better able to recognize. Puberty and pregnancy often produce increased size and pigmentation in any existing pigmented lesion (Fig. XVII), while infection, especially in relation to hair follicles, can produce progressive enlargement. The association of sebaceous cysts with hairy moles is not infrequent. However, there still remains, and will remain so long as we rely on our clinical eyes, a group of frank misdiagnoses which is significant (see Table 5). Though this group can be reduced by experience, especially regarding the diagnosis of seborrhoeic warts, there still remain two particular bugbears—angiomata (Fig. XVIII) and pigmented basal cell carcinomata (Figs.



(a) (b)

FIG. XVII. (a) Benign enlargement of an intradermal naevus during pregnancy.

(b) A radical local excision together with dissection of the axilla in continuity was unnecessarily carried out under the mistaken clinical diagnosis of malignancy.



(a)

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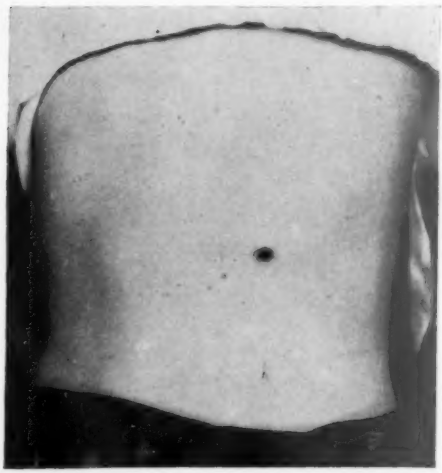


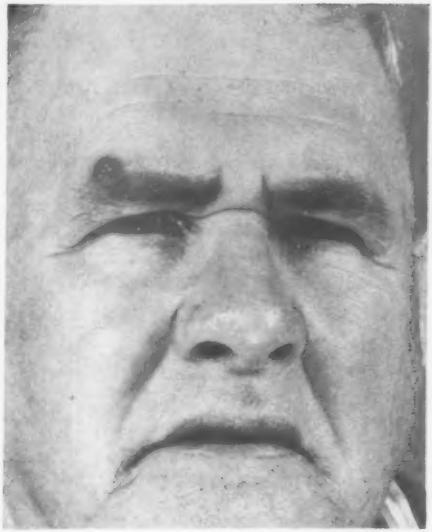
(c)

FIG. XVIII. (a), (b) and (c) Small localized angiomata with a history of recent development and increase in size. All of these lesions were mistakenly diagnosed as malignant melanomata.

XIX and XX). Even knowing of, and looking for these, we still make mistakes both ways.

In practice this means that cock-sure clinical acumen is not always good enough as a basis for proceeding with a radical destructive and mutilating operation, or a gland field clearance. All radical operations are not mutilating, however, and for the dubious small lesion (it is usually small lesions that are dubious) the removal of the extra amount of tissue which would make the excision adequate were the lesion to prove malignant, often adds little or nothing to the difficulties of repair and the resulting disfigurement. If, however, an adequate excision would involve damage to essential structures, removal of areas difficult of replacement or repair, amputations, or major dissecting operations, then we must insist on a first class pathologist at hand for confirmatory diagnosis after a reasonable local mass excision before we proceed to anything more drastic.

(a)



(b)

(c)

FIG. XIX. (a), (b) and (c) Pigmented basal celled carcinomata. All of these lesions were mistakenly diagnosed as malignant melanomata.

Prognosis and Management of Pigmented Skin Lesions

In general it now seems possible to correlate the clinical and pathological types of pigmented lesions of the skin to a point where a clinician, within a reasonable margin of error, can classify such lesions into types which have some meaning in terms of pathology, and which conjure up in the mind pictures of what they will look like under the microscope. Thus we recognize benign intradermal and combined naevi, junctional naevi, activated junctional naevi and malignant melanomas. Furthermore we are enabled to predict with some accuracy the future of any given lesion and the various possible courses which it may follow, and this in turn helps us to lay down principles of treatment.

Let us now sum up the main practical points that appear to influence both prognosis and treatment.

The most important fact to be gleaned from our own study and from these of other writers, notably Allen and Spitz, is that probably the only type of pigmented lesion which

is potentially malignant is the one which is wholely or in part composed of junctional cells, or junctional groups of cells—that is to say, either the pure junctional naevus, or the junctional component of a compound naevus which has not yet reached maturity. Although there is some evidence to show that there may be exceptions to this, nevertheless it appears to be a fairly accurate generalization.

The next important factor which influences the course and prognosis of these lesions is the overwhelming significance of puberty. Malignant melanoma prior to the onset of puberty, though not unknown, is at any rate one of the really rare surgical conditions, and here again we have to be extremely careful about the terms that we use. When we say that malignant melanoma prior to puberty is an exceedingly rare condition, our conception of malignancy is something that ultimately spreads and, if unchecked, kills the patient, or at any rate is not contained by the ordinary defence mechanisms of the body—that is to say, we use the term in a clinical sense. However, despite this assertion, there are many lesions which occur in childhood which satisfy all the common pathological criteria of malignancy, even up to the point of metastasis to lymph glands, and yet the fact is that almost without exception a diagnosis of malignancy made before puberty will not be confirmed by the clinical course of the condition. Although Allen and Spitz think that in most cases they can distinguish between the so called juvenile melanoma and the true adult malignant melanoma on microscopic grounds, we have not been able to do this in our cases with certainty. Given a certain pathological specimen which bears the criteria of malignancy, we are unable to say whether it will in fact prove malignant clinically unless we know the age of the patient. From all this, therefore, it appears that though junctional naevi, i.e., immature naevi, are very commonly seen before puberty, there seems to be some influence restraining their possible development into malignant tumours prior to the onset of puberty, except in very rare cases. It appears, too, that any lesion which appears prior to puberty and which runs its full course to maturity—i.e., which loses its junctional component and which becomes entirely intradermal in character — is a safe lesion, and thereafter has no malignant potentiality; whereas, those lesions which are not fully matured by puberty and still retain a junctional component, or which appear after puberty as either junctional or compound naevi may become malignant at any time thereafter.

FIG. XX. Multiple pigmented lesions on the face—apparently activated by recent pregnancy. The smaller lesions on the face were activated junctional naevi. The main lesion on the nose was diagnosed as a malignant melanoma, but proved to be a typical pigmented basal-celled carcinoma.

There are certain differences in structure and behaviour between congenital and acquired lesions. The life history of the mole may commence at any point from conception to the grave, but the main feature of congenital lesions which is of importance is that, because they develop *in utero* during the course of general body differentiation and growth, they are usually just part of a local abnormality which involves a number of tissues and in which pigmentation is only one

of the features of the abnormality. Such abnoral areas are not seen to develop *ab initio* after birth. In other words, the pigmented lesions that appear after birth represent an abnormality of the one group of cells in the area only, and not of all structures and tissues in the area. It appears also that the life history of the naevus cell component of congenital abnormalities is completed at an earlier stage and most of them have developed into mature naevi either by the time of birth or during childhood. Even those which do retain some junctional component in early years are almost always mature by the time of puberty, and thereafter contain no junctional element except in rare cases. Associated with these facts is the expected clinical observation that malignant melanoma only very rarely arises out of a congenital naevus.

The next important factor that deserves consideration is that at certain times during life, notably at or about puberty and during pregnancy, crops of new junctional naevi appear about the bodies of certain people and existing pigmented spots show signs of increased activity. It is noteworthy that not only may such lesions progress to maturity in the usual way, or become sidetracked into malignancy whilst there is still a junctional component existing, but the exact opposite may take place—i.e., the lesion may get smaller and even ultimately disappear on some occasions. We have been watching one patient, aged thirty, for the last three years in whom crops of typical junctional naevi have been appearing and disappearing in various parts of the body. During that period two of these lesions have become malignant while under observation and have been removed at different times. In view of such observations, it seems likely that we shall get a much clearer picture of the significance of pigmented skin lesions when we know a little more about the physiology of pigmentation, and its inter-relationship with other body functions.

Another observation which has a bearing on the course and prognosis of pigmented naevi is that junctional lesions appearing, often for the first time, in elderly people, and presenting clinically as diffuse patches of irregular pigmentation of variegated colour and indistinct outline, are particularly prone to malignant change. Microscopically, these lesions are indistinguishable from other junctional naevi seen at other times of life and probably the processes involved are similar. Nevertheless, they do form a definite clinical group, to which we have always referred as melanosis, although probably they would be better referred to as diffuse junctional naevi. This is a highly pre-malignant condition and deserves to be treated with much more attention than it usually gets in its early stages.

These, then, are the most important features which will form the background of our consideration and treatment of any given lesion at any particular age.

Treatment

There are three reasons for treating pigmented lesions of the skin—disfigurement, fear of future malignancy, and fear that the lesion may already be malignant. The first two apply before puberty, all three apply after puberty, and whatever the indications the only form of effective treatment is surgical excision.

(a) *Before puberty*, one is presented with a wide variety of pigmented skin lesions, most of which, if left to time and untouched, will reach maturity before puberty commences, but some of which will still retain a junctional component after that time. We cannot predict with any degree of accuracy which will be mature by puberty and which will still retain a junctional element at that time, except in the case of those lesions which are already existent and well developed towards maturity at birth, i.e., those usually large lesions which fall into the category of congenital malformations of an area and for which, whatever their potentialities, there is already an ample justification for treatment because of their disfiguring characteristics. These children are usually brought along to see if we can do anything to remove a disfigurement, and because we know that few, if any of them, will retain any junctional component after puberty, we need consider their treatment only in regard to disfigurement and not in regard to potential malignancy. Their treatment is excision, all in one piece or serially in stages, and we need have no fear

of cutting right through the middle of the lesion in the course of our serial excision programme. Some of the resulting wounds will be able to be sutured directly; many will require local re-arrangements of flaps and skin grafts, the details of which are not relevant to this paper.

TABLE 5

Chief clinical groups of pigmented skin lesions	*Histological diagnoses*									
	Pigmented naevi			*Malignant melanoma*	*Malformation with naevus cell*	*Other lesions*				
	Junctional naevi	*Compound naevi*	*Intradermal naevi*			*Pigmented basal cell carcinoma*	*Seborrhoeic wart*	*Pigmented papilloma*	*Angioma*	*Malformation with naevus cells*
GROUP 1 Large congenital pigmented naevi (removed for disfigurement) (62 cases)	1	42	11	1	5					2
GROUP 2 Diffuse spreading pigmented naevi in adults (melanosis) (20 cases)	12			8						
GROUP 3 Small, localized, pigmented lesions—										
A. Not suspect of malignant disease	2	16	18			2	3		2	
B. Suspect of malignant potential (96 cases)	5	18	10	8		1	8	1	2	
GROUP 4 Malignant melanoma (42 cases)	3		2	28		3		1	5	
Atypical cases				2						

In all other pigmented lesions which are presented for our consideration during childhood, we have to consider that a certain proportion of them will still remain as junctional naevi in whole or in part after puberty, and that a still smaller proportion of the whole will ultimately show malignant change later in life. We have, therefore, to decide whether it is justifiable or proper to remove all such lesions as an insurance policy against the future. Such a policy, however, if practised indiscriminately, would get completely out of hand, for there are few children who do not have a brown or black spot, or many of them somewhere around the body, and we obviously cannot remove them all. Furthermore, there are undoubtedly smaller microscopic areas of similar change which are not visible to the naked eye. We have, therefore, to compromise to some degree, and fortunately most of the cases which are presented to us that have excited attention because of their size and prominence will have the additional indication of disfigurement to justify our intervention and, indeed, in most cases, that is the parents' prime concern at the moment. We will therefore excise these lesions and in so doing we will not be afraid to cut quite close to their edges, knowing that there is no fear that they may be malignant at the time of our operation, knowing that in so doing we will satisfy the parents from the point of disfigurement, and knowing that at least one lesion will not henceforth become malignant. That then, is the picture prior to puberty, and to all intents and purposes there is no other picture during that phase of life. There is no justification whatever for most of the radical operations done for the supposed cure of supposedly malignant melanomata in childhood, irrespective of the clinical appearance and irrespective of any pathological report which may be received. It is true that exceptions to this generalization occur from time to

time, but the number of properly documented cases of malignant melanoma in childhood stands only at 18 at this time (McWhorter and Woolner, 1954), and they form only a minute fraction of the vast total of children's pigmented naevi that have come under notice over many generations throughout the world.

(b) *After puberty* the problems of treatment are very different, for after this any lesion with a junctional component may become malignant at any time before the mole reaches maturity, and here too we have to consider the lesion that is already malignant clinically when we see it. For practical purposes we can divide them all into three groups. Firstly, those that are clearly benign when we see them (Group 1); secondly, those which show some signs of activity but where we are not quite sure whether they have already transgressed the bounds of innocence or not (Group 2); and thirdly, those lesions which are obviously clinically malignant (Group 3).

Group 1

Here we have at one end of the scale the well developed mature naevus — the raised, rounded, rather pale brown mole with hairs projecting from the surface which so many women want removed from their faces for cosmetic reasons. Microscopically, these lesions are mature intradermal naevi and their potentialities for malignancy are long since past. They are simple harmless lesions which virtually never become malignant melanomata, and can be removed by incisions quite close to their margins if indicated for cosmetic reasons. Many of these show fluctuation in size and enlarge with pregnancy; such enlargement is associated with benign cellular proliferation within the dermis and must not be confused with malignant change. At the other end of the scale in this first group we have an entirely different picture, that of the flat, dark brown or black stain, flush with the surface and not associated with overgrowth of hair. It may be small and punctate or large, diffuse and irregular in outline. This is the clinical picture of the junctional naevus, and this is, in fact, the precursor to malignancy.

Now these two lesions at the two ends of the scale can be readily recognized clinically and they present no problem. The one is excised closely for cosmetic reasons and no other. The other must be excised widely because of its pre-malignant condition and because of the difficulty of determining its boundaries accurately, but there is no need to excise it deeply, for the abnormality is confined to the basal-cell layer of the epidermis. Between the typical intradermal naevus and the typical junctional naevus lies the compound or mixed lesion with some of the characters of each, often appearing as a central raised mole with a surrounding "halo" of flat pigmentation and often associated with a history of recent fluctuation in size and intensity of pigmentation. Because of their junctional component they are potentially malignant and their treatment is similar to that indicated for pure junctional naevi.

Group 2

The less experienced the surgeon, the greater the number of lesions that will fall within this category. It should be our constant aim to try and eliminate it completely as we become more experienced in diagnosis, and this has been one of the main objects of our interest in the problem. Nevertheless, there will always be some lesions so close to the borderline that we will be unable to determine clinically whether they have crossed it or not. In such cases there will be evidence of a pre-existing junctional component to the lesion, together usually with a history of increase in size, alteration in the depth of pigmentation or the appearance of a lump where one did not previously exist within a pigmented area. Where doubts exist in our minds, we should always elect to treat such cases as if they were malignant and to feel thankful for small mercies if our pathological report subsequently demonstrates their innocence. It may be said that this is too radical a view to take and that we should adopt the practice of biopsy of a limited nature to ascertain the diagnosis, and then proceed accordingly. We have had ample cause to doubt the clinical value of negative reports from limited biopsy, both in regard to this and other tumours. Furthermore, it has seemed to us that it matters little to the patient whether a small area or a large area of skin is removed provided that no irreplaceable structures are involved and that the surgeon has a complete knowledge of the available methods of replacing skin

loss. We have preferred to err on the safe side and so far have had no cause to regret such policy.

Group 3

We now pass on to the treatment of the frankly malignant melanomata. It is easy to pass this off by simply saying that the correct treatment is wide surgical excision as far as the primary growth is concerned. The word "wide" is the crux of the matter. It is all very well for a surgeon to say of a lesion on the thigh, that it should be excised with a margin of healthy skin of, perhaps, two, three, four, five or six inches in diameter according to taste, and extending down to and including an even larger area of deep fascia. But what would the same surgeon do when presented with the same lesion in the glabella region right between the eyes? Would he, in order to satisfy his arbitrary standards, exenterate both orbits in the procedure? There is nothing absurd in such a comparison. There is no particular reason to sacrifice tissue on a large scale, when such a scale is unnecessary and produces no better results than excision on a more limited scale. If we find we can remove the lesion in the glabella region without sacrificing both eyes, and without getting recurrence at the primary site over a period of many years, then clearly there is no need to excise two or three or ten times as much tissue down on the thigh. All that we, out of our experience, can say is that of 46 histologically proven malignant melanomata we have excised, we have not yet had a recurrence at the site of the primary excision (Table 6). That seems to indicate that our present policy as regards excision of the primary is adequate. Each case is assessed on its merits, and an area of excision is mapped out for each individual melanoma, without recourse to any particular rules, except for the generalizations that the bigger the lesion the wider should be the margin of excision, and that no matter how small the lesion, one should not encroach closer than half an inch from it in all directions. There are in some situations, particularly around the face, areas where even that conservative view is not practical, but it is as near as we can get to any general rule. Where the situation will allow we are often more liberal in our removal, i.e., we try to err on the side of safety. Probably the most important thing to stress is that the removal of the lesion must be planned without any consideration whatever for the method of repair that is to follow. We have had it put to us many times that is it very hard to do this and that perhaps two different surgeons should do the removal and the repair for this reason. It is a curious thing that we have never heard this extraordinary proposition put forward in relation to any other tissue than the skin. Even its most enthusiastic supporters have never been heard to suggest that one surgeon should excise the carcinoma of the colon and another should repair the bowel. There is no difficulty in planning an adequate excision for a lesion without sacrificing tissue unnecessarily, and then considering how best one can repair the defect; the two problems are quite separate and distinct and are very easily divorced from each other in one's mind. To suggest that no thought should be given to the repair until the destructive surgeon has done his worst is quite unreal and places the surgeon charged with the repair in an impossible position.

So much for the removal of the primary. What is to be done with the gland field? If the glands are clinically involved, yet operable, the issue is clear, and radical excision of the largest area of gland field practicable should be carried out. And let us not hesitate to do this, for there are many cases recorded, and we, too, have had a number, where patients have survived such operations for years (Table 6), despite an apparently hopeless prognosis at the time of operation. What shall we do when there is no clinical involvement of the regional glands? It is not possible to lay down a definite policy that will cover all cases. There is little doubt that in the case of the obviously malignant lesion, with extensive microscopic invasion of the dermis, in a person of suitable age and general health, and in a situation where the lymph drainage is clear-cut to an operable glandular field, a radical regional gland resection should be carried out forthwith, as soon as the diagnosis is confirmed pathologically. There are however other cases where there would be every justification for a wait and see policy after removal of the primary, e.g., the superficial malignant melanoma which has not yet invaded the dermis deeply and where, in fact, the conception of malignancy, although cur-

Table 6

Analysis of interim results after operation on 46 cases of cutaneous malignant melanoma, in relation to histological type and treatment

Histological Type	Nature of Operation			Interim Results																Untraced
	Excision of primary lesion only	Excision of primary and gland field		Dead or with inoperable metastases						Dead of other causes	Alive without evidence of recurrence to date after									
		Glands not involved	Glands involved	Lymphatic spread	Blood borne after						1	2	3	4	5	6	7	8 yrs.		
					1	2	3	4	5 yrs.											
A. Superficial malignant melanoma	14 cases										4	3	3	2	1	1				
B. Malignant melanoma with marked dermal invasion	18* cases	6 cases			2	1	1	1		2	5	1	1					2	2	2
			8 cases		1		1	1	1		2		1		1	2				
	Total 32 cases																			

* One of these proved inoperable at the primary operation.

rently accepted, is certainly not undeniable at this stage; or the case of the elderly person or the person otherwise not particularly fit for a radical operation; or again the lesion which is placed in a situation where removal of all possible fields of drainage would be far too radical and destructive.

The only other consideration that arises is whether the tissue lying between the primary and the glands should have anything done to it. Many policies have been elaborated in regard to this, from a complete amputation of a whole extremity, involving primary, intermediate tissue and glandular field all in one mass, to the less radical procedure of removing a wide strip of skin, subcutaneous tissue and deep fascia from primary to glands, or a more conservative policy of removing both primary and glandular field but leaving the intervening tissue. Our practice has been to confine the removal of the intervening tissue between primary and glands to those cases where the primary is so close to the regional glands that the operation can be carried out as a single block dissection of practical dimensions.

The consideration of malignant melanomata has been necessarily brief, for our prime purpose in presenting this paper has not been to discuss the treatment of obviously malignant melanomata, but rather to try to establish the behaviour and proper management of all those other common pigmented lesions which fall short of obvious malignancy. Moreover, if we are able to get some correlation between the microscopic appearance and the clinical behaviour of all these various types of lesions, then we are well on the way to an understanding which will enable us to treat them rationally and sensibly, and not in an atmosphere of ill-disguised surgical panic, based upon sheer ignorance and complete lack of understanding between surgeon and pathologist.

Acknowledgement

Most of the photography is the work of Mr. Roy Inglis—we gratefully acknowledge his skill and kind co-operation.

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GASTRIC MUCOSA WITHIN THE OESOPHAGUS

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FOR many years there has been confusion about the nature of peptic ulceration occuring in the oesophagus. It was commonly believed that such ulcers usually developed in islands of ecoptic gastric mucosa situated in the lower oesophagus. Barrett (1950) showed that such islets of gastric tissue probably did not occur, but that there was a condition in which the whole of the lower oesophagus was lined with gastric mucosa. He also made the first clear differentiation between peptic ulceration of the oesophagus and reflux oesophagitis. The position was further clarified by Allison (1953), but there are still many problems that require solution.

One of the peculiar properties of the gastric secretion is that under certain conditions it can produce ulceration of the mucosa from which it originated. If the epithelium of the duodenum or jejunum is exposed to the uninhibited action of strong gastric secretions, similar ulceration may develop. These typical peptic ulcers may develop secondary complications such as massive haemorrhage, penetration, perforation or stenosis.

When the gastric secretion acts upon a squamous epithelium such as is found in the normal oesophagus this typical peptic ulceration does not occur. Instead there is a shallow, non-penetrating ulceration with thin, yellow surface slough, slow blood loss from the surface, progressive fibrosis and stenosis.

The gastric secretion acts upon the squamous epithelium of the oesophagus under conditions which produce incompetence of the cardia. This is usually, but not invariably, associated with sliding hiatus hernia. When stenosis occurs it does so immediately above the cardia so that there is no normal oesophagus between the stomach and the line of the stenosis.

Barrett and Allison have shown that ulcers which possess all the features of peptic ulcers of the stomach do occur in the oesophagus, and that strictures of the oesophagus can occur high in the thorax with a length of apparently normal oesophagus between the stomach and the level of the stenosis. Investigation of these patients has shown that the lower part of the oesophagus is lined with typical gastric mucosa. The other coats of the oesophagus are normal. The peptic ulcers are situated in this area of gastric mucosa and they may be the site of massive bleeding, they may penetrate into the mediastinum, they may perforate into the pleural cavity or they may stenose to produce oesophageal obstruction.

FIG I. Peptic stricture of the oesophagus with a length of apparently normal oesophagus below.

At the upper end of this abnormally situated gastric epithelium there is no mechanism to prevent the flow of peptic secretions on to the normal squamous epithelium of the oesophagus. Consequently these patients are liable to develop reflux oesophagitis at this level.

Allison has named this condition "oesophagus lined with gastric mucous membrane." Some examples of this condition have been seen by Mr. C. J. Officer Brown and the author at the Alfred Hospital, Melbourne.

Case 1

Mr. W.M., aged 72 years, complained of increasing difficulty with swallowing for four years.

A barium swallow showed that there was a stricture of the oesophagus about 3 inches above the cardia with apparently normal oesophagus above and below (Fig. I). Oesophagoscopy was performed and it was said that the appearances were typical of carcinoma of the oesophagus. No biopsy was taken.

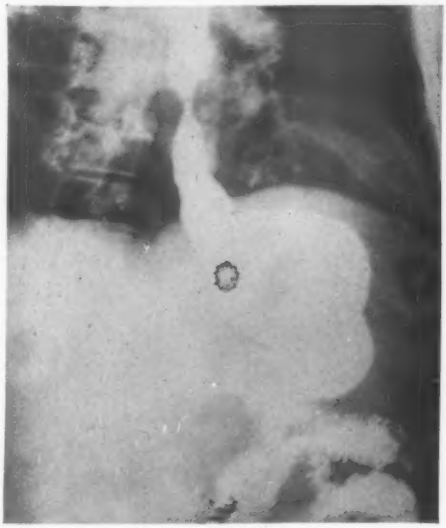

FIG. II. Stricture of the oesophagus with large ulcer crater just above the stricture.

The condition was diagnosed as carcinoma of the oesophagus. At operation a firm area in the oesophagus was felt 3 inches above the cardia. The lower half of the oesophagus and the upper half of the stomach were removed and the oesophagus was anastomosed to the stomach. Microscopic examination of the excised specimen showed that the stenosis had been caused by ulceration and fibrosis at the junction of gastric and stratified squamous epithelia.

The patient made an uneventful recovery, and now at the age of 76, four years after the operation, he is perfectly well. Since operation he has never had any difficulty with swallowing or symptoms suggestive of a recurrence of oesophagitis.

Case 2

Mr. W.Mc., aged 62 years, had had increasing difficulty with swallowing for three years. When he presented he could only swallow liquids.

Barium meal showed a stricture of the oesophagus very similar in all respects to that illustrated by last patient. The condition was diagnosed as carcinoma of the oesophagus and the patient was submitted to operation on 27th October, 1953.

FIG. III. Stricture of the oesophagus due to stenosis of a peptic ulcer in an area of gastric epithelium within the oesophagus.

When the oesophagus was exposed its external appearance was essentially normal. Some thickening could be felt at the level of the inferior pulmonary vein. In order to confirm that this thickening represented the level of the oesophageal obstruction the anaesthetist passed a large stomach tube down the oesophagus. This was held up at the site of the thickening. Instead of proceeding to a radical resection a longitudinal incision was made in the oesophagus, passing through the abnormal area. This showed that there was a dense fibrous stricture about half an inch long. Below the stricture the mucosa had the red velvety appearance typical of gastric mucosa, above was the paler squamous epithelium usually seen in the oesophagus. The strictured area of the oesophagus was excised locally and the two cut ends of the oesophagus were anastomosed to each other.

Since operation the patient has been extremely well. He can swallow normally and has had no symptoms of oesophagitis.

These two patients both had gastric mucosa lining the lower third of the oesophagus with stricture formation at the line of junction of the gastric and squamous epithelia, as the result of reflux oesophagitis.

Case 3

Mr. G.D., aged 69 years, had had increasing difficulty in swallowing for eight months. Barium meal showed a stricture of the oesophagus about 3 inches above the cardia and an appearance that suggests a large ulcer crater just above the stricture (Fig. II). Oesophagoscopy revealed a fungating mass completely filling the lumen of the oesophagus. Biopsy revealed inflammatory tissue only. Despite this it was felt that the lesion must be malignant. At operation it was apparent that there had been a great deal of peri-oesophageal inflammation but the actual site of the lesion in the oesophagus was not immediately apparent. Careful palpation revealed an area of induration about 3 inches above the cardia. The anaesthetist passed an oesophagoscope and confirmed that this indurated area represented the level of the oesophageal obstruction.

FIG. IV. Stricture of the oesophagus due to stenosis of a peptic ulcer. The ulcer niche can be seen in the stenosed area.

The lower half of the oesophagus and the upper half of the stomach were removed and the oesophagus was anastomosed to the stomach.

Microscopic examination of the excised portion showed that it contained no squamous epithelium at all. The entire length of the excised portion of the oesophagus both above and below the stricture was lined with gastric epithelium. The oesophageal obstruction had been produced by stenosis of an ulcer in this gastric mucosa.

Four months after operation the patient again complained of some difficulty with swallowing. Oesophagoscopy showed that there was some narrowing of his suture line. This was dilated and he had no further trouble of this nature. He died five years later from carcinoma of the prostate.

Case 4

Mrs. J.O., aged 64 years, had had indigestion, flatulence and regurgitation of food for many years. Over the past four months she had developed rapidly increasing dysphagia. When she presented she could only swallow a little fluid. Barium meal showed that there was a narrowing of the oesophagus about 3 inches above the cardia (Fig. III).

FIG. V. Filling defect in the middle third of the oesophagus apparently due to granulation tissue resulting from peptic inflammation in an oesophagus lined with gastric mucosa.

Oesophagoscopy showed that the stricture was situated 30 cms. from the upper alveolar margin. At 22 cms. there was a change in the character of the mucosa lining the oesophagus. Biopsy showed that the mucosa between 22 cms. and 30 cms. was of a gastric type. Above 22 cms. it was squamous epithelium. The stricture could not be dilated so it was excised and the cut ends of the oesophagus were anastomosed to each other. Microscopy showed the excised specimen to be a typical peptic ulcer with gastric mucosa above and below.

Since the operation the patient has been very well. She can swallow normally and has had no symptoms suggestive of oesophagitis.

Case 5

Mrs. I.S., aged 55 years, had never had any dyspeptic symptoms. Over three years she had noticed a gradually increasing dysphagia.

Barium swallow showed a stricture of the oesophagus in its middle third. Oesophageal biopsy showed that the mucosa just above the stricture was of a gastric type. The stricture was dilated very easily and the patient has maintained the patency of the oesophagus by swallowing a mercury filled rubber bougie daily.

Barium meal carried out after the dilatation shows a small ulcer niche within the narrowed portion of the oesophagus (Fig. IV). The radiologist made every attempt to demonstrate the presence of an associated hiatus hernia or oesophageal reflux, but was unable to do so.

FIG. VI. The same patient as Fig. V. Barium swallow normal three years after the examination illustrated by Fig. V.

Cases 4, 5 and 6 show examples of stenosis of the oesophagus which has resulted from true peptic ulcers, situated in gastric mucosa lining the lower half of the oesophagus. This is a different pathological process to that in the first two patients in whom the stenosis was due to reflux oesophagitis.

Case 6

Mr. A.T., aged 75 years, had had indigestion for many years. In 1949 he developed a marked degree of oesophageal obstruction over a few months. Barium meal at that time showed a filling defect in the oesophagus with obstruction (Fig. V). The condition was apparently diagnosed as inoperable carcinoma of the oesophagus and no treatment was carried out.

A barium meal three years later showed no abnormality (Fig. VI). A few months later the patient had a massive haematemesis and melaena and nearly died. After a further four months he again developed marked oesophageal obstruction, this time at a higher level than his original lesion (Fig. VII). Oesophageal reflux could be demonstrated, but there was no hiatus hernia. Oesophagoscopy showed the oesophagus to be blocked by a fungating mass. Biopsy yielded only inflammatory tissue.

FIG. VII. The same patient as Fig. V and Fig. VI. Stricture of upper third of oesophagus. This examination was made four months after that illustrated by Fig. VI.

He was seen by us for the first time at this juncture. We dilated the stricture and taught the patient to swallow a mercury filled rubber bougie daily. The patient has remained well on this regime for two years. Recently we re-investigated his case and biopsy of the mucosa just above the level of the stricture yielded gastric epithelium.

Case 7

Mrs. P.W., aged 62 years, had had indigestion and vomiting after meals for thirty years. In 1949 a barium meal showed the presence of a large hiatus hernia but there was no oesophageal obstruction. Two years later she complained of increasing difficulty in swallowing and barium meal at this time showed a marked narrowing of the lower end

of the oesophagus (Fig. VIII). This stricture was treated by dilatation with partial relief of her symptoms.

A barium meal one year later showed that there was now no obstruction at the lower end of the oesophagus but that the oesophagus was narrowed at the level of the arch of the aorta (Fig. IX).

FIG. VIII. Case 7. Barium swallow showing stricture formation just above the cardia.

Oesophagoscopy showed that the stricture was 22 cms. from the upper alveolar margin. Biopsy of the mucosa at this level yielded gastric epithelium. As it was not possible to dilate the stricture it was decided to excise it.

At operation the external appearance of the oesophagus was normal. In order to help identify the level of the obstruction the anaesthetist passed a large stomach tube. This was held up at the level of the arch of the aorta, but a little further pressure caused it to pass beyond the stricture. We felt that in view of this fact we would not proceed to excise the stricture, but that we would treat it by dilatation as it had been so readily dilated this time, despite previous failures. The patient was a poor operative risk, and we were glad of the opportunity to avoid a major procedure. We repaired the hiatus hernia and closed the chest.

Three days after operation the patient died as the result of a cerebral vascular thrombosis.

At autopsy it was shown that the upper one-third of the oesophagus was lined with squamous epithelium. The lower two-thirds was lined with gastric epithelium. The stomach was normal in its size and position (Fig. X).

Cases 6 and 7 are similar to the first two, in that the oesophageal stenosis occurred as the result of reflux oesophagitis, but in these instances the stricture was much higher. They also illustrate an amazing variation in the site and degree of their stenosis at different times.

FIG. IX. Same patient as Fig. VIII. Examination made one year later. There is now a marked narrowing of the oesophagus just below the level of the clavicle.

At this point I would like to summarize some of the features of these cases. The average age was 65 years. Their clinical histories varied from a few months to 30 years. There was an associated hiatus hernia in three cases, two others had no hernia, one of these had oesophageal reflux, the others did not. There is no evidence re hiatus hernia in the remaining two cases. At operation it was often difficult to locate the site of oesophageal obstruction, the external appearances of the oesophagus and stomach were relatively normal.

Any theory which attempts to explain the nature and origin of this condition must be able to encompass all these facts.

It has been said that the condition is of congenital origin, the oesophagus having been lined by gastric epithelium throughout life. If this condition is congenital in origin then there must be a second factor which only comes into play in the second half of the patient's life, for that is when the complications become apparent.

FIG. X. Autopsy specimen from Case 7. Note the white squamous epithelium in the upper one-third of the oesophagus and the dark gastric type epithelium in the lower two-thirds.

Allison (1953) has put forward the rather ingenious theory that the condition may rest basically upon an incompetence of the cardia which may or may not have its origin in a hiatus hernia. He visualizes that a patient with an incompetent cardia has the lower end of the oesophagus constantly bathed in acid gastric secretions. If this produces oesophagitis with destruction of squamous epithelium, any consequent healing in the presence of acid gastric secretions might occur more readily by an up growth of gastric epithelium rather than by a down growth of squamous epithelium. If this is true, one can imagine a sheet of gastric epithelium creeping up the oesophagus and taking over areas that have been denuded of squamous epithelium by gastric secretions which came originally from the stomach, but which might be contributed to later by the secretions from the new areas of gastric mucosa in the oesophagus.

This theory is very attractive as it would explain the late onset of the symptoms which could not develop until the patient had first developed a hiatus hernia in the fifties.

The theory, however, has no basis on clinical or experimental fact and it might well collapse when it is investigated more closely. In one of our cases no oesophageal reflux could be demonstrated despite careful examination. This is contrary to Allison's view.

Several of the earlier cases were submitted to operation under the diagnosis of carcinoma of the oesophagus. Now that we are aware of the syndrome of the "oesophagus lined with gastric mucous membrane" we are less likely to make this mistake, but we might be exposing ourselves to the more serious error of delaying operation in patients who are suffering from oesophageal malignancy, by regarding them as examples of peptic stricture.

Unless one has complete understanding of the nature of a condition it is not possible to lay down sound principles for its treatment. It is because of this lack of complete understanding that we are not in a position to be dogmatic about the correct treatment for the oesophagus lined with gastric mucosa.

Some of the strictures that are produced by either form of this condition can be dilated quite readily. The patency of the oesophagus can then be assured if a mercury filled rubber bougie is swallowed each day. Two of our patients have been treated in this manner.

If the stricture cannot be dilated easily then it must be excised. This raises two problems. How much oesophagus shall be excised? What method shall be used to restore the continuity of the alimentary tract?

It appears to be illogical to restore continuity by a method which will expose the squamous epithelium of the oesophagus to the action of gastric secretions. This would be likely to lead to further reflux oesophagitis and recurrent stricture formation. It, therefore, follows that all oesophagus which is lined by gastric mucosa must be excised, and the remaining portion should be anastomosed to the jejunum, never to the stomach. Allison states that this is the correct approach to the problem and he advocates an oesophago-gastrectomy with oesophago-jejunal anastomosis in all cases.

There is no doubt that this is theoretically sound, but such an operation is a very big procedure for a non-malignant condition in a patient who is approaching seventy years of age, and who has been the victim of oesophageal obstruction for some months. There can be no doubt that in a large series of cases there would be a significant mortality that would offset the advantages which the procedure may have over other less heroic lines of treatment.

In two of our early cases we carried out an oesophago-gastrectomy with oesophago-gastric anastomosis in the belief that we were dealing with carcinoma of the oesophagus, and the result in both instances was very satisfactory.

Because of this experience we decided to attempt an even more restricted procedure and in two cases we simply excised the stricture locally, and anastomosed the two cut ends of the oesophagus. This is a very easy operation that only takes about one-third of the time necessary to do an oesophago-gastrectomy with oesophago-jejunal anastomosis.

These patients have only been followed for twelve months but so far the results have been excellent. They have had no symptoms suggestive of oesophagitis. In one of the patients oesophagoscopy was performed three months after his operation and there was no evidence of oesophagitis.

When one divides the oesophagus it is usual to carry out a vagotomy at the same time because of the intimate relationship of the vagus nerves to the oesophagus. It is possible that this may have some bearing on the fact that those patients have remained free of the oesophagitis which one would expect to develop.

SUMMARY

There is a clinical state in which the lower end of the oesophagus is lined by gastric epithelium. The origin of this gastric mucosa is a matter of conjecture. It may be complicated by the development of reflux oesophagitis and high oesophageal stricture or by the formation of typical peptic ulcers which may haemorrhage, perforate or stenose. The correct line of treatment of this condition is a matter of debate but we have been pleased with the results achieved by a conservative approach to the problem.

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HYPOTHERMIA

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THIS study introduces the subject of hypothermia for discussion, briefly mentioning the early pioneer concept for cancer treatment, the subsequent revival of interest in clinical research laboratories, the more recent applications to cardiovascular surgery, and at the same time taking the opportunity to present a preliminary report on our own work. Physiological data has been verified, experience has been gained in producing the hypothermic state, in defining safe limits of temperature, in handling experimental animals, and finally the knowledge so gleaned has been employed to study some basic problems of intra-cardiac surgery.

The state of hibernation by which certain animals are able to protect themselves from severe winter conditions by deep sleep, the body temperature falling to a degree only a little above that of the surrounding atmosphere, and living entirely upon body reserves accumulated during the warm season, yet prompted to rouse if the temperature falls dangerously towards freezing level, has received scientific interest throughout historical times. It is known that hibernating animals will survive temperatures of 38°F (3°C) without shivering and without harm, whereas in non-hibernating animals shivering is violent and death occurs between 68°F (20°C) and 59°F (15°C). The matter was given close attention fifteen years ago in the belief that reduction of body temperatures, although harmless to adult differentiated tissue, including the nervous system, was lethal to embryonic malignant cells in the same way as deep X-rays. An investigation was undertaken in human cases of advanced hopeless cancer (Smith, 1940), the temperature being reduced to between 40 and 50 degrees Fahrenheit for some days at a time. Most cases withstood the ordeal without complaint and with relief of pain, but there was no permanent curative effect which could not better be attained by other means. In five cases there was death from sudden unexplained myocardial failure and there was significant mortality from pneumonia beyond that usually attributable to advanced malignancy.

The possibility that hypothermia might be used to extend the scope of surgery and supplement anaesthesia, was carefully investigated and reported in a series of papers by Bigelow (1950) and a research team at Toronto. By artificially cooling anaesthetized dogs from the normal of 100.4°F (38°C) to 68°F (20°C) and re-warming them rapidly in a water bath after the experiment, it was shown that metabolism fell to 18 per cent. of the original value at 69°F, with a comparative diminution of oxygen requirements. The physiological changes, largely physical in nature, were not harmful to the most delicate tissue and, on restoration of normal temperature, were shown to be completely reversible without an oxygen debt. Shivering was controlled by the anaesthetic until sleep narcosis supervened at 83°F (28°C), but the slowness and inefficiency of breathing at this low temperature made provision of positive pressure insufflation necessary.

Death from cardiac crisis took place between below 59°F (15°C) and frequently above 69°F (20°C). The cause of this has engaged the closest study. The effect of hypothermia on the heart, minimal to begin with, was observed to become progressively more pronounced and serious with the falling of temperature. Bradycardia with slowing of conduction and inversion of the T wave was followed by some degree of heart block and accompanied by hyper-irritability of the myocardium and frequently by spontaneous ventricular tachycardia and ventricular fibrillation. The hypothermic state was proven to give protection to the brain with the circulation arrested. In a series of experiments (Bigelow, 1950) 15 per cent. of dogs survived arrest for fifteen minutes with the atrium opened, a state previously incompatible with life. Of the 75 per cent. mortality two-thirds died immediately on restoring the circulation either from ventricular fibrillation or, less

commonly from cardiac arrest. Of the survivors 60 per cent. died after twelve hours from shock, the remainder recovering completely after a few days of lethargy. From the discussion on this classic work it appeared likely that intra-cellular electrolyte changes were responsible for these failures (Moore). The harmless nature of hypothermia on cells in the absence of arrest was supported by the record of growth and division of mammalian tissue cultures at 83°F (28°C) for six weeks (Firor, 1950). Both cardiac irregularity and post-operative shock may have been due in part to carbondioxide build-up with lowering of the pH and altering of the electrolyte balance resulting from the method of positive pressure insufflation used in the experiment.

In the hypothermic state at 80°F, with the circulation arrested for twelve minutes, there was recovery in 84 per cent. of dogs (Cookson *et alii*, 1952) whereas normothermic animals suffered death or grave neurological damage after nine minutes arrest (Gibbon). From these experiments it was concluded that a temperature of 80°F was less dangerous and prolonged nervous tissue survival by 50 per cent. as compared with normothermic animals.

A subsequent paper (Bigelow and McBirnie, 1953) confirmed a suspected tendency to ventricular fibrillation in hypothermic dogs as compared with primates. Of twelve monkeys cooled from 100°F (38°C) to 68°F (20°C) in half an hour, eleven survived complete circulatory arrest for from fifteen to twenty minutes and were active and well co-ordinated within two hours. It was shown that the ground hog, induced into a hibernating state by cooling, could survive complete circulatory arrest for one hour at 38°F (3°C) with complete recovery except for some decreased resistance to infection. In hibernating animals there is more efficient nerve conduction at low temperatures compared with non-hibernating animals and this may have some bearing in comparing the different behaviour of the heart at these temperatures.

Newly born mammals survive body temperatures fatal to adults of the same species and it has been ascertained that the rat and the cat at birth survive 34°F (1°C) and 45°F (18°C) temperatures respectively, whereas in adults death occurs below 63°F (17°C) and 65°F (18°C).

The protective effect of lowered body temperature on the tissues is generally accepted. It has been shown conclusively that the brain is protected during the hypothermic state. Electroencephalographic waves, constant as far down as 68°F (20°C), disappear below this but reappear on re-warming. Clinically there is no reduction of intelligence following complete circulatory arrest in monkeys for from fifteen to twenty minutes and this has been our experience in sheep and pigs.

The occurrence of air embolism as a precipitating factor in ventricular fibrillation and arrest, already well known and experienced by surgeons opening the great veins of the neck or thorax, presents grave hazards in the performance of intra-cardiac operations with the heart beating, especially when the septum is opened, for air sucked into the ventricles is delivered to the coronary arteries, either after circulation through the lungs or directly from the left ventricle. This problem is by no means solved but by occlusion of the great veins and allowing the ventricular chambers to empty, immediate air embolism is prevented when the heart is opened. Because the auriculo-ventricular valves remain open, and the semilunar valves remain permanently occluded during circulatory arrest (Swan, 1953), air embolism during intra-cardiac manipulations does not occur until the circulation is released and air trapped in the chambers is carried onwards. This can be prevented (Swan, 1953) by flooding the open auricle beneath normal saline and allowing all bubbles to disperse before the atrium is closed and the great veins released. If air embolism has occurred cardiac massage after closure and release, by restoring the cardiac circulation, may be efficacious in reviving the beat, as has been experienced in human operations at normal temperatures.

The problem of spontaneous ventricular fibrillation remains largely unsolved although electrolyte changes well known to affect the heart, have been demonstrated in the blood stream after circulatory arrest. A report of the successful application of this basic knowledge (Swan, 1953) is an indication of ultimate solution.

It is well known that the following factors are likely to produce ventricular fibrillation in the excessively irritable ventricular myocardium in the hypothermic state.

1. Diminished oxygenation of the blood and the giving of intravenous adrenalin.
2. Local cardiac irritation as by cardiac catheter, ventricular incision or pulling on the auricular septum.
3. Venous distension (Bigelow) indicating over-filling of the heart between beats.
4. Release of the circulation after cardiac arrest.

The first two causes can be eliminated. Over-distension of the great veins, indicating over-filling of the heart, is guarded against by the very slow release of the circulation, the superior vena cava first, followed by the inferior vena cava; however, spontaneous fibrillation is not eliminated by these precautions. Intra-cellular electrolyte changes of fundamental importance in the normal heart beat, mirrored to some extent in the blood stream, at present are considered the most likely cause of this state. Oxygen consumption by the beating heart is shown to be fairly normal. It is measured by the arteriovenous difference between coronary arterial and coronary sinus blood flowing during cardiac arrest under hypothermia (Penrod, 1951). It is probable that it may be necessary to supply oxygen requirements of the heart during arrest by perfusion of the coronary arteries with oxygenated blood.

It is known that excess of potassium ions will stop the heart in diastole and that excess of calcium ions will provoke ventricular fibrillation and arrest the heart in systole. Restoration of the normal heart beat from a state of ventricular fibrillation has been attained by this method (Hooker, 1929). Potassium ions, to which the hypothermic heart is peculiarly sensitive, and which have been shown to precipitate ventricular fibrillation experimentally (Elliot and Crismon, 1947), were shown to accumulate in the body serum during cardiac arrest with precisely the same result on resumption of the circulation (Swan, 1953). The selective action of calcium and potassium on the heart has been put forward as providing a means of defibrillating the heart in hypothermia. With the heart filled with saline and the aorta clamped beyond the coronary artery origin, defibrillation was accomplished (Swan, 1953) by injection into the aorta proximal to the occlusion of first potassium chloride solution (5 milliequivalents 2 cc. at a time) to stop fibrillation, and then calcium chloride (2 cc. of 2 per cent. at a time) to initiate the heart beat, the drugs being delivered through the coronary circulation by cardiac massage. It was reported possible to restore normal rhythm beginning first in the ventricle itself, and later moving to the sinus node, in seven of eight hypothermic animals (Swan, 1953).

There is experimental proof of the grave danger of ventricular fibrillation following sudden changes from high carbon dioxide and low pH in the blood to normal (Brown and Miller, 1952). In circulatory arrest, due to continued cellular metabolism there is an alteration of electrolyte balance between cells and serum, with excess potassium ions in the serum, and tissue carbon dioxide build-up causing a lowering of the pH, the severity of this depending on the duration of arrest and the degree of hypothermia. Venous blood in hypothermia is highly oxygenated and has a normal carbon dioxide content. It remains thus in the great venous reservoirs during circulatory arrest. Experimental investigation of the possibility that the sudden change of medium on release of the circulation might be a factor in precipitating ventricular fibrillation was undertaken by comparing the survival rate in two groups of hypothermic dogs, in one of which the circulation had been arrested in the ordinary way, and the other upon which hyperventilation had been performed to wash out the carbon dioxide and raise the pH before the circulation was stopped. The fibrillation rate was reported as falling from 50 per cent. to 8.3 per cent. in comparing the two groups (Swan, 1953).

The knowledge of prevention and cure of cardiac crisis, gleaned from so many sources, was now considered safe enough for clinical trial of hypothermia in man to provide protection to the central nervous system after artificial circulatory arrest, during which intra-cardiac operations of considerable physiological moment were carried out.

Some reservations are reported concerning the restoration of normal rate and rhythm

once severe sinus bradycardia and ventricular tachycardia have supervened and in this connection intermittent intra-arterial perfusion with oxygenated blood is recommended as a prophylactic measure (Juvenelle *et alii*, 1954), made continuous and combined with rapid re-warming after the onset of ventricular fibrillation.

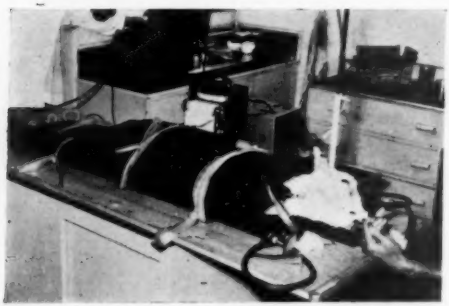

FIG. I. Hypothermia being induced in a lamb. The jugulo-carotid area is exposed, an intravenous drip is inserted into a cannulated leg vein, the animal wrapped in a plastic blanket within which water at 3°C circulates. The temperature is recorded, and the heart examined by a direct writing electrocardiograph. Intermittent positive pressure insufflation is maintained with oxygen as shown, by mechanical means.

Experimental method

Closely shorn sheep or weaner pigs, fasting for twenty-four hours before operation, were anaesthetized with intra-peritoneal sagatal and lightly curarized. Oxygenation was maintained by an endo-tracheal tube connected to an oxygen cylinder with a short rubber tube fitted with a T-way connection or a breathing valve which permitted intermittent positive pressure pulmonary inflation (Fig. I). Observations were made on the pulse rate, the blood pressure, general metabolism and the physiological changes in the blood cells, and electrolyte balances, *etcetera*, at 7°F intervals. The pulse rate and the heart beat were recorded by a direct writing electrocardiograph. The arterial and venous blood pressures were ascertained by electromanometric means after inserting a needle into the exposed carotid artery and jugular vein respectively. Cardiac output was ascertained with a catheter in the right atrium using the Fick principle.

Experimental observations

Each animal showed a progressive fall in body temperature and respiratory rate. At 88°F (31°C) cold narcosis was apparent and spontaneous respiration gradually ceased. In preliminary experiments animals were taken down to find the fatal level of temperature for each particular animal, this being between 76°F (24°C) and 79°F (26°C) in lambs and between 59°F (15°C) and 69°F (20°C) in pigs, death occurring suddenly from ventricular fibrillation or cardiac arrest.

In the next series of experiments the temperature was kept above this dangerous level, physiological measurements were recorded and the animals rapidly re-warmed and allowed to recover. On recovery there was no apparent alteration in the mental or physical state of the animal apart from lethargy for twenty-four hours.

There was a progressive fall of blood pressure and heart rate as the temperature fell with a comparative rise on re-warming (Fig. II).

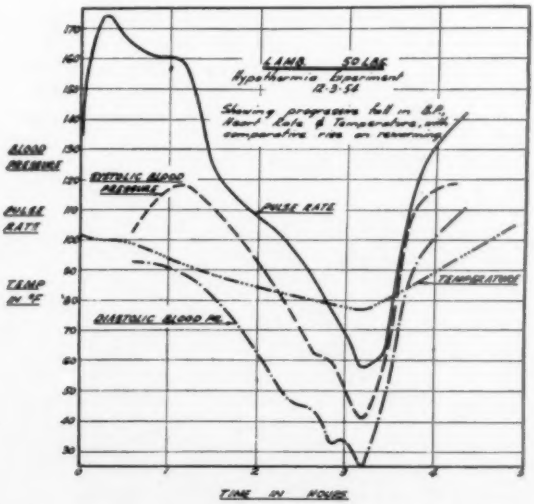

FIG. II. Showing the progressive falling of blood pressure and heart rate with comparative rise on re-warming.

It was found important to control shivering and to give artificial respiration with pure oxygen during induction of the hypothermic state, for without such control the pulse was shown to fluctuate, the temperature to fall slowly, and oxygenation not to be fully maintained (Fig. III).

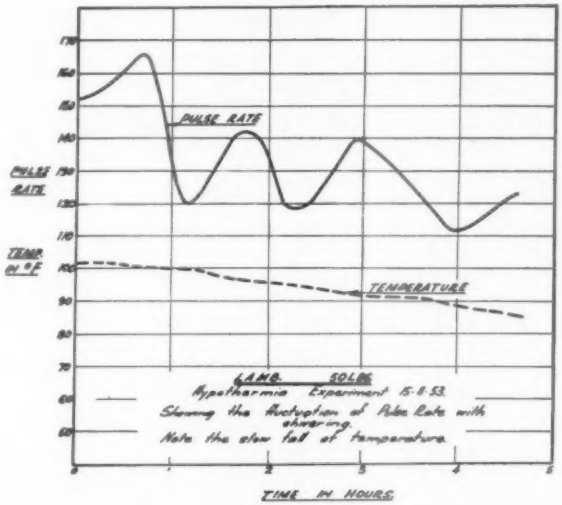

FIG. III. Effects of shivering showing fluctuations in pulse rate, impairment of oxygenation and difficulty with induction of hypothermia.

There was intense vasoconstriction in the peripheral vessels and the venous pressure was noticed to rise, with depression of the heart rate, but there was no significant degree of haemoconcentration.

The cardiac output showed a gradual fall during cooling and a rise on re-warming, being in the lamb about 4 litres per minute at 101°F (38°C), falling to 3.2 litres per minute at 80°F (27°C) with a similar rise on re-warming.

The metabolism was shown to fall progressively with the temperature, being 20 per cent. to 30 per cent. of normal at 83°F (28°C) in the lamb (Fig. IV).

The blood picture was shown to remain remarkably normal. There was no haemolysis, no significant haemoconcentration, some progressive depression of the white blood cells and some diminution of the arteriovenous oxygen difference consistent with diminished metabolism (Fig. V). There was a slight increase in sodium ions and potassium at lower temperatures. Electrocardiographically there was progressive sinus bradycardia with prolongation of conduction time (PR interval) and delay in repolarization of the ventricular muscle (QT complexes). The T wave was shown to be increased in amplitude at lower temperatures and there was a tendency toward the development of ventricular tachycardia from possible aberrant ventricular foci which, however, were observed usually to revert to normal sinus rhythm. At lower temperatures the sinus bradycardia was shown to progress quite suddenly with gross prolongation of conduction, increase of the QRS complexes and flattened, diphasic or reversed T waves, and was recognized as a warning of cardiac crisis with impending ventricular fibrillation or cardiac arrest. This grave bradycardia could be relieved almost immediately with intravenous atropine, reducing vagotonia. The progressive changes of cooling were reversible on re-warming except for grave bradycardia and ventricular fibrillation which usually progressed to cardiac arrest (Fig. VI).

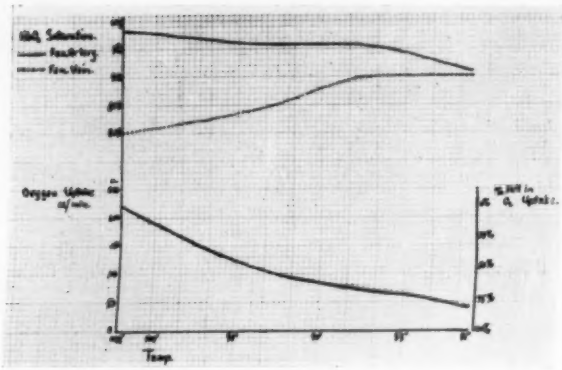

FIG. IV. Effect of hypothermia on oxygen uptake and arterial and venous oxygen content.

during circulatory arrest was usually successfully restored by release of the clamps and oxygenation of the blood. Ventricular tachycardia was shown to revert to sinus rhythm under this treatment. It was not prevented by intravenous injection of Procardyl. Arrhythmia was best forestalled by preliminary hyperventilation, adjustment of the temperature above the known critical level and restoration of the circulation as soon as arrhythmia threatened. There seemed to be no protective value in calcium and it was hard to assess the value of intravenous digoxin.

Arrest of the circulation during hypothermia

The chest was opened through the 4th intercostal space on the right side, widely spread, the azygos vein divided between ligatures, tapes being passed round the venae cavae, the pericardium opened vertically and a purse string suture inserted into the auricle and controlled loosely by the Rumel tourniquet. The lung was hyperventilated for several minutes and the venae cavae clamped, all subsequent procedures and responses being recorded on the direct writing electrocardiograph. Congestion of the venae cavae was a constant finding and became more obvious after arrest of the circulation. Most animals survived clamping of the venae cavae for nine minutes without neurological damage and one sheep survived occlusion for twenty minutes at 79°F (27°C). Severe bradycardia

In our limited experience, under hypothermia, ventricular fibrillation occurring during or following surgery with the circulation arrested, has been fatal in all cases despite electro-convulsive shock and attempts at defibrillation with intra-arterial potassium and calcium.

Intra-cardiac surgery under hypothermia (serial electrocardiographic record, Fig. VII)

The great veins were clamped and, allowing a period of one minute for emptying of the heart, the atrium was opened inside the purse-string suture and four traction sutures inserted, two at either extremity of the incision. It was found wise to fill the chest with saline, to close the atrium, and to let

Temp. (F.)	102°	95°	88°	81°
(A) HAEMATOLOGY.				
Hb.	10·3 Gm.	11·3 Gm.	13·2 Gm.	12·8 Gm.
W.B.C.	13,200/cmm.	8,400/cmm.	5,600/cmm.	4,000/cmm.
Diff. Polymorphs	47%	45%	68%	62%
Eosmophils	1%	2%	2%	2%
Lymphocytes	52%	53%	30%	36%
E.S.R.	0 mm.	0 mm.	0 mm.	0 mm.
Platelets	331,000/cmm.		412,000/cmm.	412,000/cmm.
Clotting Time	7½ mins.		7½ mins.	6½ mins.
(B) BIOCHEMISTRY.				
Serum Bilirubin	0·2 mgm%	0·2 mgm%	0·2 mgm%	0·2 mgm%
Sodium	334 mgm%	337 mgm%	370 mgm%	395 mgm%
Potassium	9·4 mgm%	10·5 mgm%	10·0 mgm%	9·7 mgm%
(C) O_2 and CO_2 EXCHANGE.				
O_2 Consumption	217 cc/min	121 cc/min	81 cc/min	45 cc/min
Arterial HbO_2 Satn.	98%	96%	96%	91%
Venous HbO_2 Satn. (Fem. Vn)	80%	83%	90%	90%
Arterial CO_2 Content	59 vols.%	57 vols.%	57 vols.%	54 vols.%
Arterial pH.	7·85	7·86	7·90	

FIG. V. Some physiological responses to hypothermia.

the circulation resume with re-oxygenation of the tissues on completion of this manoeuvre, and then, after an interval, to arrest the circulation once again, opening the atrium widely for surgical manipulation.

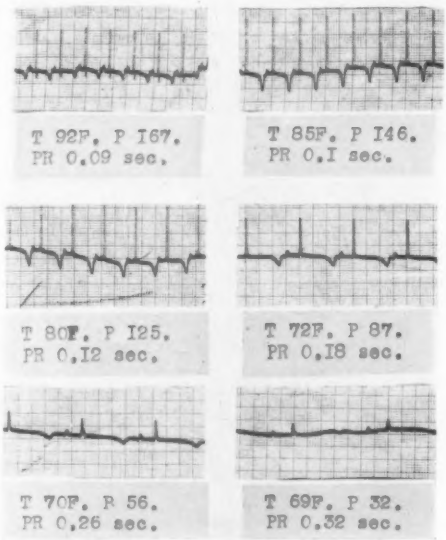

FIG. VI. Electrocardiographic records of a pig undergoing experimental hypothermia.

The following observations were made:

1. *Exposure*

There was diminution in size of the heart. Considerable blood was found to pass directly into the ventricle and obscure the interior of the atrium. It was readily controlled with a suction nozzle in the coronary sinus which manoeuvre had no effect on the heart. The AV ring was seen to contract down with ventricular systole and the flimsy valves to be mostly closed and blood not regurgitating from the ventricle. With considerable practice, and using the coronary sinus for a landmark, the anatomy of the atrium could be visualized.

2. *Rhythm*

Shortly after opening the atrium changes in rhythm were noticed to occur, commencing often with ventricular tachycardia. Bradycardia was most ominous and likely to be followed within seconds by ventricular fibrillation. Between two and three minutes (usually less) was the longest single period at which manipulations could be performed without the onset of alarming changes. By the recognition of bradycardia and the taking of immediate measures to deal with this, it was possible to restore normal rhythm. This was done by flooding the chest with saline, closing the atrium by the purse-string tourniquet, releasing the clamps, and ventilating the lung with pure oxygen. The sequence of progressive depression of the nodal rhythmicity, depression of conduction through the Purkinje system and eventually grave sinus bradycardia, interpolated by frequent spontaneous bursts of ventricular tachycardia and finally ventricular fibrillation after a short interval, was characteristic in all experiments. It was shown that the safe interval for clamping the circulation (speaking of the heart beat only) was longer in the hypothermic animal but the response eventually was much the same. Atropine, gr. 1/50th, injected into the right ventricle was shown to change sinus bradycardia to tachycardia within thirty seconds. With the restoration of normal rhythm the clamps were re-applied, artificial respiration stopped and the atrium re-opened and this could be repeated on several occasions. It was possible to restore the heart from grave bradycardia by releasing the circulation with full oxygenation of the blood ensured by intermittent positive pressure insufflation with pure oxygen and the administration of atropine as already described which abolished vagal tone. In our experience the sequence described, once arisen, was liable to recur with decreasing voltage of ventricular beats and recurrence of sinus bradycardia, followed inevitably by irreversible ventricular fibrillation within a few beats. It has not been possible in animals to restore permanently the normal beat after ventricular fibrillation has supervened, either by electro-convulsive shock or chemical means. There has been some temporary restoration of the beat using an electrode inserted down the jugular vein to the region of the sinus node as an artificial pacemaker. The purse string was essential for closing the atrium rapidly and allowing resumption of the circulation quickly in dealing with impending crisis.

3. *Manipulation*

(1) Disturbances to rhythm on insertion of the purse-string suture were shown to be transitory and to vanish immediately these manipulations were completed.

(2) There was no disturbance on incision of the auricle and none on catheterizing the coronary sinus.

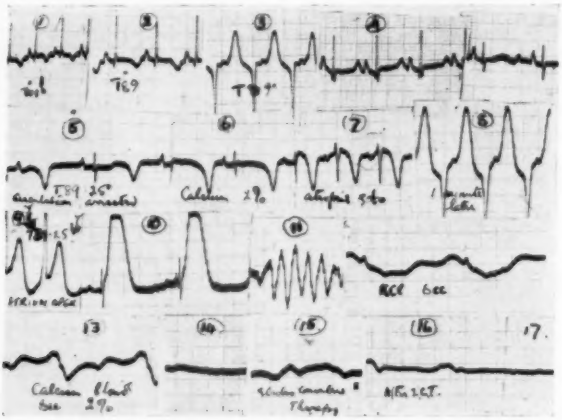

FIG. VII. Serial electrocardiographic record of a pig undergoing a cardiac operation with the circulation arrested. In No. 2 the usual sinus bradycardia is developing, suddenly interrupted by venticular tachycardia which reverted spontaneously in 4. In 5, after arrest of the circulation, the T wave becomes progressively depressed and the heart slows, calcium chloride 2% having no effect 6, but atropine being followed immediately by sinus tachycardia 7 and ventricular tachycardia 8. On opening the atrium 9, grave sinus bradycardia ensued 10, passing to ventricular fibrillation 11, which was not cured by potassium chloride or calcium chloride and passed to arrest 14. The normal rhythm was only temporarily restored by electro-convulsive therapy 16.

(3) There was severe arrhythmia and often ventricular fibrillation following traction on the atrial septum or insertion of the finger through the atrio-ventricular valve.

(4) It was found necessary to have co-ordinated teamwork with two assistants, wide exposure of the interior by traction sutures, and a good suction fitting into the coronary sinus. Provided the coronary sinus (just medial to the entry of the inferior vena cava) was catheterized, the limbus of the septum could be visualized about one centimetre above and behind this and an incision or suture of the septum carried out. It has proven no easy matter to provide a good view in the cramped surroundings, even with experience, and operative disasters have occurred. Incisions too far forward have resulted in perforation of the medial wall of the atrium at the root of the great vessels or if too low have caused damage to the AV node in front of the coronary sinus with resultant heart block. Damage has been done to the medial cusp of the tricuspid valve causing death from acute tricuspid regurgitation and in some cases complete incision of the septum has not been accomplished.

(5) Death has occurred from coronary air embolism resulting in irreversible ventricular fibrillation on several cases in which septal defects have been made with the heart open. By emptying the heart before the auricle is opened, further circulation through the great vessels is arrested until the circulation is resumed. The closure of the atrium beneath saline after air has escaped from the heart has allowed the resumption of circulation with the heart cavities filled with fluid and without fear of air embolism (Swan, 1953).

Surgical applications of hypothermia

During operations to correct Fallot's Tetrad both in babies and in children, the safety and protective value of mild hypothermia has been shown to reduce the incidence of cerebral damage from anoxia during anaesthesia from 13 per cent. to 2½ per cent. by cooling to between 93 and 96 degrees Fahrenheit. The temperature during operation, before cooling was adopted, was discovered to register between 100 and 102 degrees Fahrenheit in 60 per cent. of cases (Potts *et alii*, 1952). This principle has undergone further development for direct heart operations on pulmonary infundibular and valvular stenosis. It has been our experience in this laboratory that direct intracardiac surgery with the heart arrested is a dangerous procedure at present unsuitable for routine human application. The liability to crisis is considered to outweigh protection afforded to the central nervous system.

There is general agreement that the prognosis for most atrial septal defects is good. It seems that there will not be medical support for operative closure until the mortality approximates to that of ligation of a patent ductus. This achievement may be possible by progress in controlling ventricular fibrillation, maintaining a cardio-cerebral circulation, or perhaps by development of indirect methods not involving the arrest of the circulation.

There has been so much satisfaction with the Blalock operation and with direct operations for pulmonary stenosis (Brock) that more radical treatment with the circulation arrested will have to be shown more successful without adding to the operative risk.

It has recently been reported (Delorme, 1952) that the hypothermic state anticipates the late effects of surgical shock by preventing tissue hypoxia and enzyme activity. General acceptance of this concept would necessitate a radical alteration of treatment.

The recently discovered variation of thyroid gland activity with depression and restoration of temperature may be a clinical application in hyperthyroidism.

Arterial grafting of the thoracic aorta (Beattie *et alii*, 1953), previously impossible owing to the danger of spinal cord damage following more than thirty minutes of circulatory arrest, has been accomplished in the laboratory with safety. Application for protecting the brain can be foreseen in cerebral vascular surgery and more remotely in the treatment of severe cerebral injury.

SUMMARY

1. A short review of hypothermic development has been attempted.
2. Physiological data obtained from pigs and sheep undergoing hypothermia have been discussed.
3. Experience on these animals with hypothermia in intra-cardiac surgery has been presented.
4. Clinical applications are briefly summarized.

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VOLVULUS OF THE SMALL INTESTINE WITH A NORMAL MESENTERY

A REVIEW OF THIRTY-NINE CASES

By Thomas F. Rose

The Royal North Shore Hospital, Sydney

Introduction

WHILST a long mesentery with a short base and a redundant loop of bowel are the predisposing causes of volvulus of any part of the colon, these are uncommon factors in volvulus of the small bowel, except, of course, in the newborn where volvulus of the midgut occurs when there is non-rotation of bowel associated with a long mesentery whose base is the narrow duodeno-colic isthmus.

Volvulus of the small bowel is therefore almost always found in persons with a normal mesentery and no redundant loop of bowel, and thus is quite a different entity from large bowel volvulus.

It is the purpose of this article to discuss 39 such cases of small bowel volvulus.

Incidence

In a series of 210 cases of acute mechanical small bowel obstruction due to causes other than a strangulated hernia seen at the Royal North Shore Hospital of Sydney in the past eleven years, there were thirty-seven (17.6 per cent.) cases of volvulus of the small intestine, and in each instance the mesentery and its attachment were normal. Thirty-one cases involved portion of the ileum, five portion of the jejunum, and one all the small bowel (Footnote *).

An additional two ileal cases were seen and treated elsewhere (Footnote **).

The 17.6 per cent. incidence of volvulus in this series of acute mechanical small bowel obstruction was higher than that usually reported in similar series from the United States of America where it varies from 1.3 to 4 per cent. (Wangansteen, 1946; Bollinger and Fowler, 1953; Drugas and Schiff, 1954).

A different story comes from Africa and India, but, as pointed out by Dickson (1950), the African series of Sartorius (1947), in which there was an incidence of 27.3 per cent. of volvulus in acute small bowel obstruction in the Bantu, is not comparable with Western figures because it referred to healthy young men few of whom had ever had previous abdominal operations likely to cause the adhesions which are a common cause of volvulus. Ohja (1950) from India found 18 cases in 47 small bowel obstructions, again none due to post-operative adhesions (Footnote ***).

Causation of Volvulus of the Small Intestine

Predisposing causes

Many causes of small intestine volvulus have been mentioned in the different reports of this condition and they appear to differ

Footnote * — Two other cases of ileal volvulus occurred at the Royal North Shore Hospital during this time but are excluded because, though in each instance the mesentery was anatomically normal, in one (previously reported by Mitchell [1954, Case 2]) the predisposing cause was a large lipoma of the mesentery, and the other occurred in the sac of an inguinal hernia.

Footnote ** — Nine of this series were personal cases. Six were ileal, one of which has already been reported by Mitchell (1954, Case 1) and one by Rose (1953, Case 2). Three were jejunal, two of which were reported by Rose (1954).

Four others, one jejunal and three ileal, were seen in consultation.

The remainder, including one jejunal, were abstracted from the records of the Royal North Shore Hospital.

Footnote *** — In an interesting article from North Bihar, India, Banerji (1950) stated that he found 309 cases of small bowel volvulus in 394 cases of mechanical small bowel obstruction, a truly astonishing figure of 83 per cent. However, Banerji describes a strange condition of folding of the mesentery rather than a volvulus, so there is no need for its further consideration here.

according to race and country. Most of them, even after dismissing mesenteric anomalies which, by definition, are excluded from this series, are not applicable here, but they make an interesting study nevertheless.

Firstly, it must be remembered that the small bowel volvulus of the poorer classes of India and Africa appears to differ from that of white races. In the former, it commonly affects the greater part of the small bowel and is hence accompanied by marked distension. In Ojha's (1950) Indian series of 18 cases, 10 had all the small intestine involved. Hamman (1951) pointed out that this is a commonplace finding in small bowel volvulus in the Bantu of Africa. On the other hand, in white races, only a comparatively short length of intestine is affected (Bailey, 1954).

The cause of this difference is thought to be the nature of the diet. In India and Africa, the diet of the poorer classes who suffer from volvulus is farinaceous and bulky, badly and incompletely cooked, and hastily bolted as the one main meal of the day.

Dickson (1950), supported by post-mortem measurements, made the statement that, probably in consequence of such a diet, the small intestine of the Bantu is longer than that of the European, and he thought that this might account for the frequency of small intestine volvulus in that race. In Eastern Europe, such a diet is said to cause redundancy of the pelvic colon though why in one race the small bowel is involved and in the the other, the large, is a matter for speculation.

In any case, in India and Africa, the diet is said to cause chronic small bowel distension which is known to be a cause of volvulus (Kerr and Kirkaldy-Willis [1946] writing from Kenya; McWatters [1945], Ojha [1950] and Banerji [1950] writing from India). On the other hand, Dickson (1950), investigating radiologically young male Bantus, mixed barium with their food and thought he found some evidence of hypermotility of the intestine but the results were inconclusive.

Coming back to Western countries, as in this present series, where dietetic factors are not a consideration in the aetiology, adhesions, whether congenital or acquired, are the commonest predisposing cause. A Meckel's diverticulum may be associated with a congenital adhesion to the umbilicus (the remnants of the vitello-intestinal canal), or acquired adhesions when the diverticulum is inflamed, both of which types can help form a volvulus of the ileum.

A tumour hanging from the antemesenteric border of the bowel may be a predisposing cause (Aird, 1949).

Exciting causes

When an exciting cause is obvious, the main one is an upset in normal bowel motility. This may be hypermotility due to a purgative (Tagart, 1950), lead poisoning (Berger and Lindberg, 1951), foreign bodies in the bowel lumen (Ripstein and Miller, 1950), ascariasis (Kafarov, 1949) or intussusception (Young, 1951). On the other hand, gaseous distension as in a mechanical obstruction (McKechnie, 1951) or that due to steatorrhoea (Avery Jones, 1952) is also an exciting cause.

Miliary tuberculosis affecting the bowel has been blamed (Dupin, Delahousse and Lacroix, 1951) but the mechanism is obscure.

The exciting cause may be found in pregnancy where the expanding uterus, by stretching any adhesions which may be present, can cause a small bowel volvulus (Bellingham, Mackay and Winston, 1949). In the same way, removal of a large abdominal tumour with the consequent alteration of bowel position and any existing adhesions may cause a small bowel volvulus.

In spite of these varied causes, it must be realized that we do not yet know the exact mechanism of volvulus formation especially in those persons with normal anatomy. Why, for instance, does a purgative cause only increased peristalsis in millions of people whereas every now and then it will cause a volvulus?

Then, again, many cases of volvulus, clinically indistinguishable from those with definite predisposing causes and with or without exciting causes, have no apparent cause at all.

SMALL BOWEL VOLVULUS IN ANIMALS

If we venture into the field of veterinary medicine to seek an explanation of the cause of small bowel volvulus we are as much in the dark as ever.

Volvulus of the small intestine is widely distributed throughout domestic animals. It is most commonly found in the horse (Hutyra, Marek and Manninger, 1938), but one cannot compare volvulus of the small bowel in the horse with that in the human with a normal mesentery, because in the horse the small bowel is on a long mesentery and its contents are always bulky, a condition similar to that in human pelvic colon volvulus, or perhaps the small bowel volvulus of Eastern and African races. It is not similar to the small bowel volvulus of the European with a normal mesentery.

In those animals such as the cat and dog, where the mesentery is comparatively short, small bowel volvulus is rare but does occur and is comparable to that in this series. Hobday (1953) mentioned the case of a dog in which all the small bowel was involved. A recognized predisposing cause in dogs are adhesions following abdominal operations which have been followed in some instances by recurrent volvulus. Kirk (1939) cited chronic constipation as a cause of volvulus in adult cats and dogs.

However, there is still here no explanation of why a condition almost perfectly normal in some individuals (chronic constipation) will cause volvulus in others.

In the field of zoological medicine, volvulus is rarely seen. Sir Edward Hallstrom, President of the Taronga Park Zoological Gardens, stated that there has never been a case there. An instance is reported in a young mink and in an elephant but details are lacking (Hutyra, Marek and Manninger, 1938).

CAUSATION IN THIS PRESENT SERIES

Predisposing causes

The cases in this series were able to be divided into three groups according to the presence or absence of a predisposing cause.

Group 1

This was the so-called idiopathic type in which no predisposing cause was found. This is a rare form of volvulus according to Ficarra (1953) although the number of cases in this series alone leads one to doubt this.

Sixteen cases belonged to this group, 15 ileal and one total small bowel (less than 70 cases of this latter have previously been reported according to Joyeux and Courty [1953] but they did not review the Indian or African literature where examples are more common than in the European or American).

Group 2

The predisposing cause was a single band of probable developmental origin running from the antemesenteric border of the apex of the involved loop of bowel to the posterior abdominal wall in 5 instances and the anterior in three.

There were 8 cases in this group, 5 ileal and 3 jejunal (Footnote ****).

Group 3

The predisposing cause was due to acquired adhesions.

Apart from adhesions matting loops of bowel together, in all instances except one the apex of the involved loop of bowel was attached by adhesions to some relatively fixed point, for instance, the abdominal scar or the uterus or broad ligament. In the one exception, a loop of jejunum had slipped through an adhesion fixed by both its ends to the posterior abdominal wall and then twisted itself into a volvulus. This is somewhat analogous to those patients with rents in their mesentery through which a loop of bowel slips and then twists. It is also especially common in the horse.

There were 15 cases in this group, 13 involving the ileum and 2 the jejunum.

Six were due to adhesions following removal of an inflamed appendix, 5 of which were also gangrenous and ruptured with local

Footnote **** — None of the patients in Groups 1 or 2 had had any prior abdominal operation except one in Group 1 where an appendicectomy had been mistakenly performed for a previous similar attack, and one in Group 2, a jejunal volvulus due to an obviously old fibrous band well away from an area of acute peritonitis complicating a recently removed appendix.

peritonitis. The volvulus occurred at intervals varying from eighteen hours to fifteen years after appendicectomy. In 3 instances, the appendix had been removed through a paramedian incision, and in each of these, all some years after appendicectomy, the cause was the adherence of the involved loop to the scar. The one jejunal case in this post-appendicectomy group is interesting in that it occurred six years after appendicectomy for an inflamed unruptured appendix situated in the right iliac fossa and removed through a McBurney's incision. Convalescence was normal, yet at operation for the volvulus, there were old widespread abdominal adhesions. This is of interest in that it confirms observations that widespread abdominal adhesions may occur following a localized focus of infection probably because of the spread of inflammatory fluid between the coils of bowel.

The other jejunal volvulus in Group 3 occurred six weeks after a Wertheim hysterectomy complicated by a pelvic abscess.

Two cases were due to adhesions following small bowel resection for irreducible intussusception two years and six years previously when aged thirteen year and seven months respectively.

One was due to plastic adhesions caused by a co-existent tubo-ovarian abscess to which the involved loop was attached.

One was due to adhesions following a retroperitoneal haemorrhage (Rose, 1953).

One was due to neoplastic adhesions due to a carcinoid tumour of the ileum.

Three were due to plastic adhesions caused by an inflamed Meckel's diverticulum on the apex of the involved loop, one occurring in an adult of 54 years and two in infants. McKechnie (1951) reported two such patients aged 70 and 77 years respectively. Christensen (1954) stated that volvulus of a Meckel's diverticulum, with or without the loop of bowel to which it is attached, is extremely rare.

Thus, not only may late post-operative or inflammatory adhesions cause a volvulus, but so may early plastic adhesions in the convalescent period (Turner and Sloan, 1951), though there were only two in this group, one eighteen hours post-appendicectomy and one six weeks after a Wertheim hysterectomy. Cave (1953) also reported cases occurring in the immediate post-operative period but not related to the actual operation.

Exciting causes

In the 16 cases of Group 1, in addition to there being no predisposing causes, in only one was there an apparent exciting cause, in a man who took a purgative (castor oil) and promptly had an acute ileal volvulus which made him collapse with shock.

In the 8 cases of Group 2, though there was a definite predisposing cause for the volvulus, in only one, a patient with a jejunal volvulus, was there an obvious exciting cause, namely a partial obstructive distension of all the small bowel from recent adhesions.

In the 15 cases of Group 3, all with predisposing causes, in only three was there an obvious exciting cause. In the first, a female patient had a jejunal volvulus sixteen days after a hysterectomy for a very large fibroid whose fundus reached into the epigastrium. This resulted in a change of position of the loops of bowel which were the subject of adhesions following removal of an inflamed appendix six years previously. In the second, a carcinoid tumour with its neoplastic adhesions caused chronic low partial small bowel obstruction with gross distension both above and below the site of volvulus for three weeks prior to an ileal volvulus occurring. In the third, generalized small bowel distension was due to a pelvic abscess and adhesions six weeks after a Wertheim hysterectomy. In addition an ileal fistula had been present which then closed spontaneously. This led to even greater distension and within two days a jejunal volvulus occurred.

Thus, of the 39 patients in this series of small bowel volvulus, 23 only (Groups 2 and 3) had a predisposing cause and 5 only (one each in Groups 1 and 2, and three in Group 3) had an obvious exciting cause. Thus, 15 patients had neither predisposing nor apparent exciting causes. This is quite a different picture from that of the series of McKechnie and Priestley (1936) where in only three of 37 operations on 34 patients could no predisposing cause be demonstrated,

the others being caused mainly by adhesions. (They also included cases with a long mesentery, malrotation of gut and exomphalos which, however, have been deliberately excluded from this present series.) McKechnie and Priestley's series is comparable to Moritz and Moreton's (1950) series where in only 3 of 36 cases was there no predisposing cause.

Sex

There were only 5 females in this series, one in Group 2 and four in Group 3. In McKechnie's series (1951) there were 16 females and 14 males.

Age

In Group 1, the ages varied from 2 to 72 years, four being over seventy years.

In Group 2, the ages varied from 13 to 40 years, and in Group 3, from 6 to 79 years, excepting those with a Meckel's diverticulum where the ages were one, one and a half, and 54 years.

History of Previous Attacks

Five patients (one in Group 1, and the others in Group 3) had attacks of volvulus formation which spontaneously subsided prior to the one requiring operation. Three of these patients had one prior attack only, two months to two years prior to the one requiring operation. One even had a laparotomy just after such an attack and, nothing abnormal being found, a normal appendix was removed. The fourth had 4 attacks similar to the one eventually requiring operation in the previous six years and the fifth had 6 attacks in the previous two years.

In the remaining 34 patients whose one and only attack was the one requiring operation, there were 6 patients whose attacks commenced slowly in a subacute fashion, the symptoms waxing and waning in severity but eventually increasing in intensity until they became acute and constant. This period lasted from twelve to seventy-two hours, and, as the bowel was still viable at operation, it was obvious that the volvulus was only twisting slowly and loosely and possibly untwisting as well until it finally became tight causing the attack to become markedly acute. Other patients whose volvulus twisted rapidly and tightly from the start, in similar lengths of time developed gangrene of the affected bowel.

In the other 28 patients, the attack commenced acutely from the commencement with no amelioration of symptoms until operation was performed.

In other words, the symptomatology depended on the progress of the lesion; whether the twist was slow or fast, and whether it was tight or loose. A sudden quickly tightening volvulus was associated with severe pain and shock and early gangrene of bowel, usually within a matter of hours. In a slow, loose volvulus, the pain was less severe, there was little shock, and the bowel was often viable even after days of illness.

In the quick type the mortality was higher than in the slow, mainly because a few hours delay in diagnosis in the former was lethal because of the consequent delay in operation, whereas in the latter the same delay was not so dangerous.

Symptomatology

The symptoms and signs attributable to the volvulus itself were more or less the same in all groups. They were superimposed upon those due to any predisposing or exciting causes when present.

Pain

The primary symptom was pain, and in acute cases its severity was one of the striking characteristics of this lesion, helping to differentiate it from other causes of intestinal obstruction.

The character of this pain varied. In a quickly twisting, tight volvulus, the pain was severe and continuous from the start, almost solely due to the involvement of the mesentery. In the looser, more slowly twisting type of volvulus, the pain was less severe and waxed and waned as the volvulus tightened and relaxed. The pain here mimiced a colic from its intermittency. In this type a true colic could also occur from the increased peristalsis of the obstructed gut above the volvulus. As the volvulus tightened, the pain would finally become severe and continuous.

The position of the initial pain varied with the position of the volvulus and the length of the affected bowel. When the jejunum was affected, the pain radiated from the epigastrium to the left side of the abdomen.

When the ileum was affected, it commenced similarly and radiated to the right side of the abdomen, especially to the right iliac fossa. In one instance, however, the pain in a long jejunal volvulus was found on both sides in the upper abdomen, whereas in a long ileal volvulus, the pain was on both sides in the lower abdomen. Occasionally in a long volvulus, whatever its position, the pain was generalized all over the abdomen. In one ileal volvulus, the initial pain commenced in both loins before radiating all over the abdomen. In no patient did the pain radiate through to the back as might be expected when the root of the mesentery twisted.

In those patients in whom the volvulus became gangrenous, the pain eased, though not entirely, as the base of the mesentery, though twisted, was still viable and sensitive. However, the generalized pain of peritonitis would then supervene.

Shock

This again depended on the speed and severity of the volvulus. Shock in these cases was due to two causes. Firstly, in the sudden cases, there was a neurogenic type of shock which caused a sudden collapse due to impulses from the twisted mesentery. Secondly, there was a true oligaemic type of shock due to the tremendous loss of bloodstained fluid into the peritoneal cavity. So much actual blood was lost in this transudate, that intraperitoneal haemorrhage was diagnosed in at least one case here. The resultant picture of shock in these cases was a summation of these two types. Later in those cases in which peritonitis, either pre-operative or post-operative, developed, peripheral circulatory collapse often eventuated. In 3 of the jejunal cases, shock was made worse by the further loss of fluid and electrolytes due to the copious vomiting of the high obstruction.

Excluding the three infants in this series, in the other 36 patients (whose ages ranged from six to seventy-nine years), the severity of the pain was such that in 8 instances shock markedly dominated the pre-operative clinical picture. In six of these eight cases, the shock started with the initial pain, whilst in the other two it came later as the mesentery tightened. In 6 of these, in spite of apparently adequate resuscitative measures, this shock-like state lasted throughout and after operation, so much so that two (one being the patient with the volvulus of the whole of the small intestine) died post-operatively in a state of shock, whilst the other 4 took forty-eight hours to recover from the shock.

In one other severe case, shock did not manifest itself until after operation when it lasted three days almost with the death of the patient. It was noticed in some instances that the state of shock was often accentuated after the volvulus was untwisted even though adequate resuscitative measures were being used at the time.

The diagnosis of volvulus in the 3 infants (one in Group 1 and the two in Group 3 due to an inflamed Meckel's diverticulum) was first suspected when each collapsed with shock so severe it caused one to die post-operatively. Unfortunately, in infants who cannot yet talk, pain may be very difficult to diagnose so that it is well to remember the aphorism that a sudden state of shock in a young child is an abdominal catastrophe until proved otherwise.

Thus, of the twelve patients with severe shock, three died of the shock itself.

Vomiting

Vomiting often occurred with the pain at the onset of the volvulus due to the twisting of the mesentery. In the slower cases, it sometimes recurred with the onset of each pain. Later, vomiting was due to the mechanical obstruction and was especially early and copious in the high obstruction of a jejunal volvulus. Later still, vomiting was due to the peritonitis consequent upon rupture or gangrene of the involved bowel.

Tenderness

Because volvulus is a strangulation-obstruction, tenderness was an early sign even in the slow cases. It was first localized to the region overlying the volvulus and then became widespread even before peritonitis supervened.

Rigidity

This was only in evidence when rupture and peritonitis occurred.

Distension

Distension was always present. In early cases, it was slight and localized to the area of the volvulus. Later, as the volvulus tightened and the affected loop of bowel became tense, a tender cystic tumour could sometimes be felt there. This occurred in 7 cases, including the one reported by Mitchell (1954). Six of these were ileal and the tumours were found in the right iliac fossa: one was a long jejunal loop and the tumour was found in the epigastrium and right hypochondrium. Generalized distension due to the mechanical obstruction proximal to the volvulus was uncommon as death or operation usually occured first. If peritonitis supervencd, the distension due to paralytic ileus occurred.

In 3 cases, generalized small bowel distension was present prior to the volvulus occurring and, in fact, was the exciting cause. It was present both above and below the volvulus.

Borborygmi

Borborygmi were well marked in many cases especially the slower ones.

Bleeding per rectum

Only in one instance did bleeding per rectum occur and as a consequence an intussusception was mimiced. This was also the only patient who had diarrhoea, all the others being constipated except in three instances in which the bowel action was normal early in the illness though no flatus was passed.

"Sign of relief"

Evans and Bigger (1947) stated that these patients are uncomfortable when supine and seek a position of relief which avoids traction on the mesentery. Tagart (1950) described his "sign of relief" by which his patient eased the pain by supporting his prone trunk clear of the bed on his elbows and knees.

However, the sign of Tagart was not seen in this series, the characteristic position of these patients being to lie supine, at times with the hips flexed.

Radiography

The abdomen of 16 of the later patients in this series was studied radiologically. Early in the illness, fluid levels localized to the position of the volvulus were demonstrated. These were high and to the left in one case of jejunal volvulus and high and to the right in another jejunal volvulus where the involved loop was at least three feet long and lying transversely with apex in the right hypochondrium. The fluid levels were low and to the right in most cases of early ileal volvulus.

In later cases, levels became more generalized as the bowel proximal to the volvulus becamc obstructed. These pictures also showed the dilated coils of small gut proximal to the volvulus.

In those three cases following pre-existing generalized small bowel obstruction, as might be expected generalized fluid levels were present before the volvulus occurred.

The diagnostic method of abdominal tap performed by Leonard and Derow (1937) and Thompson and Brown (1954) to demonstrate the presence of bloodstained transudate was not used in this series. Though these authors say it is without danger, nevertheless it is a blind procedure and quite unnecessary.

DIAGNOSIS

In early cases, in most instances, the diagnosis of an intestinal obstruction, even that it was a strangulation-obstruction, was able to be made, though the exact cause was not elucidated pre-operatively except in one instance only in a late post-appendectomy case in Group 3. In three instances of the slower loosely twisting type of ileal volvulus, the obstruction was only partial for quite some time so that each patient had normal bowel actions. Consequently, the diagnosis of the obstructive element was missed and acute appendicitis diagnosed, though the pain of volvulus is usually more severe than that of appendicitis. On the other hand, in two young male patients, generalized abdominal pain, caused by a large obstructed appendix filled with pus under high tension, was so severe and accompanied by screaming and shock that a volvulus was diagnosed.

In the children, intussusception was diagnosed even though bleeding per rectum did not occur. This was also the pre-operative diagnosis in the adult who passed blood per rectum.

One patient was diagnosed initially as an intraperitoneal haemorrhage thought to be a complication of a recent hysterectomy. The true picture became apparent shortly afterwards after blood was administered and the lesion progressed. Hamman (1951) pointed out that the presence of signs of blood loss (due to the bloodstained transudate) and the finding of a cystic tumour (the volvulus) on vaginal examination could make one diagnose an ectopic gestation or haemorrhage into an ovarian cyst.

In the 7 later cases where a tender cystic tumour was palpable, the correct diagnosis was made pre-operatively in all.

In the later cases still, where peritonitis had supervened, this was correctly diagnosed but the cause was not even suspected.

Thus in this series, 8 cases only were diagnosed correctly before operation.

TREATMENT

Owing to the loss of fluid into the abdominal cavity containing protein and blood cells, the elements of shock and blood loss were present in a combined form so that in many instances it was necessary to administer whole blood. Where shock alone predominated plasma was given, though in more recent cases the plasma expander, dextroven, was used. Human albumin was not used in this series as it has only just become available. It will be used in future as its advantage is that it carries no risk of serum hepatitis.

Whatever parenteral therapy was used, this plus gastric suction was continued after operation until shock was overcome and bowel function had returned.

As soon as fluid balance was reasonably established, abdominal exploration was performed.

OPERATIVE FINDINGS

These varied according to the stage of formation of the volvulus and the presence of any predisposing or exciting cause.

There was always copious bloodstained free fluid present in the abdominal cavity even in early cases. (It is interesting to note that Weber [1934] once found chyle peritonitis in a case of small bowel volvulus in an infant.)

The volvulus was twisted in a clockwise direction except in 2 instances of jejunal volvulus when it was twisted in an anticlockwise direction (Rose, 1954). The degree of twist varied from 180 to 360 degrees or more.

In each instance the mesentery and its base were normal, a finding confirmed in those patients who later came to autopsy.

The length of the involved bowel varied from a few inches in infants to total small bowel in one adult. Usually in was from one to three or four feet. The bowel looked as does any strangulated bowel, being red and oedematous with extravasated blood in the mesentery and bowel in early viable cases in tight strangulations and even in late cases in loose twists. In later cases still, gangrene was present and in these rupture of the bowel had often occurred resulting in peritonitis.

In the three cases with an inflamed Meckel's diverticulum, this was acutely inflamed and its tip was attached to the anterior abdominal wall with recent adhesions. The base of the diverticulum was attached to the apex of the involved loop of bowel. There was no twist in the diverticulum itself, the volvulus involving the loop of bowel only. In one instance the bowel was still viable but in the other two the bowel and diverticulum were gangrenous and peritonitis was already present.

OPERATIVE TECHNIQUE

If there was no cause as in Group 1, the viable volvulus was merely untwisted. That this was sufficient is shown in the follow-up where no recurrence was found to have occurred. Resection of a loop of viable bowel, as has been suggested to prevent a possible recurrence, would be a very drastic measure in these sick patients, especially when recurrence, if it does occur, may affect an entirely different loop of bowel. In this series, there were actually no recurrences.

When the volvulus was gangrenous, resection and anastomosis of the involved loop was performed.

In Group 2, the offending band was divided and the volvulus untwisted or resected according to its viability.

In Group 3, the cause was dealt with and the volvulus again untwisted or resected according to its viability.

70 years with a twelve hour history with the whole of the small bowel involved. The other was aged 48 years with a seventy-two hour history.

Ten patients had gangrenous bowel. In 7, resection was performed. Four of these

TABLE 1

SUMMARY OF MORTALITY

Operation	No. of Cases	No. of Deaths	Cause of Deaths
Untwisting of viable volvulus only (Group 1).	6	2	Continuing shock.
Untwisting of viable volvulus with division of a congenital adhesion (Group 2, including three jejunal).	7	0	
Untwisting of viable volvulus with division of acquired adhesions (Group 3, including one jejunal with drainage of a pelvic abscess).	10	1	Pre-existing abdominal sepsis and ileal fistula.
Untwisting of an apparently viable jejunal volvulus with division of acquired adhesions (Group 3).	1	1	Peritonitis due to perforation of bowel.
Untwisting of viable volvulus with resection of Meckel's diverticulum.	1	1	Continuing shock.
Resection of gangrenous volvulus and Meckel's diverticulum.	1	1	Continuing peritonitis.
Resection of gangrenous volvulus with primary anastomosis (7 cases in Group 1, one in Group 2).	8	4	Pulmonary embolus in one; continuing peritonitis in one; peritonitis from leaking anastomosis in two.
Resection of carcinoid of ileum with gangrenous volvulus.	1	0	
Enterostomy for gangrenous volvulus.	1	1	Continuing peritonitis.
No operation (moribund on admission) including two Group 1, and one Meckel's, all with gangrenous volvulus.	3	3	Continuing peritonitis.
	39	14	

MORTALITY

This was high, 14 patients in this series of 39 dying (36 per cent.). The deaths included two with a jejunal volvulus and the one with all the small bowel involved.

Group 1

Six patients had viable bowel which was untwisted. Two of these died, both of shock commencing before operation. One was aged

survived, one losing five feet of ileum. The three who died were aged 13, 45 and 69 years, the latter losing seven feet of ileum. Death was due to peritonitis in two due to the leakage of the anastomosis, and to a pulmonary embolus on the fifth day in the third. One patient, aged 50 years, was thought to be too ill for anything but a jejunostomy and died of peritonitis. Two, aged 48 and 54 years, were moribund from

peritonitis on admission and died without operation.

Autopsy in these deaths never showed any cause for the volvulus either predisposing or exciting.

Thus, 8 of the 16 in this group died (50 per cent.).

Group 2

Seven patients in this group had viable bowel which only had to be untwisted and the adhesion cut. All survived.

One patient, aged 35 years and with a thirty-six hour history, had a gangrenous volvulus already with peritonitis on admission. Resection was performed but death ensued from continuing peritonitis.

Thus one in 8 died (12½ per cent.).

Group 3

Of the 3 patients with a volvulus due to an inflamed Meckel's diverticulum, all died. Of these, one adult male, aged 54 years, with a thirty-six hour history, died of general peritonitis due to gangrene of the diverticulum and its loop of ileum before operation could be performed. One twelve-months-old child with a twenty-four hour history died of general peritonitis after resection of the gangrenous diverticulum and loop of ileum. The other child, aged eighteen months, with a twelve hour history, died of shock which commenced before operation in which an inflamed Meckel's diverticulum was removed and the viable volvulus merely untwisted.

Ten of the other 12 cases had viable bowel and simply required division of adhesions and untwisting of the volvulus, one in addition having a pelvic abscess drained. This latter patient died, not from the results of her untwisted viable jejunal volvulus but from the prolonged results of pre-existing sepsis and an ileal fistula. This was confirmed by autopsy where the involved loop was viable and the volvulus had not recurred.

One patient, whose bowel was thought to be viable at operation after a fifteen hour jejunal volvulus, died six days later from peritonitis from a small perforation in the affected loop of bowel. Autopsy disclosed this small perforation on the mesenteric border in an otherwise recovered bowel.

One patient who had a gangrenous volvulus due to a carcinoid tumour with resection of three feet of ileum recovered.

Thus of the 15 cases in this group, 5 died (33.3 per cent.).

This mortality, though high, compares favourably with those of similar series. Moritz and Morton (1950) had 12 deaths in 36 cases. McKechnie (1951) had a 46 per cent. mortality but many were late cases. Leonard and Derow (1938) had 9 deaths in 16 cases (56 per cent.), 7 of these dead patients requiring more extensive surgery than simple untwisting.

Ojha (1950) in 18 cases had 12 deaths (66 per cent.) but in his series, there was an average of three days between the onset of the illness and operation.

CAUSES OF THE HIGH MORTALITY IN THIS SERIES

In most instances, the cause of the high mortality was related to the time taken to diagnose the urgency of the condition by the referring doctor. As this time lengthened, the mortality rose due to the increased incidence of gangrene. It is indeed interesting and puzzling to note the long time it took most of these patients to be hospitalized because the difficulty in exact diagnosis is really no excuse for delaying hospitalization as the patients all obviously had an "acute abdomen" requiring operation.

In the 16 cases of Group 1, the length of time from the commencement of symptoms to operation varied from six hours to four days (in 15 cases it was over twelve hours); in the 8 cases of Group 2, in only 2 was it under eight hours, varying in the others from eight to twenty-four hours; in the 15 cases of Group 3, 6 were operated on under eight hours and 9 over eight hours (up to two days) after symptoms commenced. (In Group 3, diagnosis appeared to be easier, probably because of the presence of a previous abdominal scar. It was certainly not expeditious in those with a Meckel's diverticulum.)

In addition to the time factor, the type of volvulus was important. The mortality of the fast, tightly twisting was much higher

than that of the slow, loosely twisting type again because of the quicker incidence of gangrene in the former.

The factor of age was not important except for the special difficulty in diagnosis in the case of infants, when at the commencement of the illness, an "acute abdomen" was not even suspected.

Whilst the overall mortality in this series is high, encouragement may be gained if the series is split into the first 25 cases and the last 14 cases seen in the past four years.

In the first 25 cases, there were 12 deaths. Thirteen patients had gangrenous bowel. Nine of these died, 5 after resection, one after an enterostomy only, and 3 without operation as they were moribund on admission. The other three deaths, in patients whose bowel was viable, were due to shock.

In the last 14 cases, there were 2 deaths only. Only one patient had an obviously gangrenous bowel and this patient survived resection. One patient, whose bowel was thought to be viable, died from a perforation after a simple untwisting. The other death was due not to the volvulus but to pre-existing abdominal sepsis.

The cause of these improved figures was not so much earlier diagnosis (as even here quite a proportion were not operated on for twenty-four hours after the illness commenced, in fact some not for three to four days), but there was a greater proportion of the slower, more loosely twisting types, thus accounting for the lessened incidence of gangrene.

Improved anaesthesia and pre-operative and post-operative treatment, including blood transfusions, undoubtedly played their part as well.

FOLLOW-UP

Of the 25 survivors, 5 were unable to be traced. Four others were operated on within the past four months and are too recent for follow-up. All have been so far well.

Yet 3 others have died from other causes but had had no recurrence of the volvulus in the two to five years they had survived operation. One of these had a viable ileal volvulus untwisted at the age of 65 years. This was due to adhesions from an appendicectomy fifteen years previously. She then developed an acute ileal obstruction twelve months after her volvulus operation due to more adhesions. She recovered from this but died of a cerebral haemorrhage six months later still.

The remaining thirteen patients (5 in Group 1, 3 in Group 2 and 5 in Group 3) were well with no evidence of recurrence in from one to eleven years.

That recurrence of a volvulus is a hazard is shown by the series of McKechnie and Priestley (1936) where the volvulus recurred in two of 31 survivors, in one instance twice.

SUMMARY

A series of 39 cases of small intestine volvulus occurring in bowel with a normal mesentery is discussed. Thirty-three involved the ileum, 5 the jejunum and one the whole of the small bowel.

They were able to be divided into three groups according to the presence or not of a predisposing cause. In Group 1, consisting of 16 cases, there was no apparent cause at all. In Group 2, with 8 cases, the predisposing cause was an old, tough fibrous band running from the apex of the involved loop of bowel to the abdominal wall. In the 15 cases of Group 3, the predisposing cause was due to acquired adhesions, either inflammatory or post-operative, and in one instance neoplastic.

Symptoms and signs are discussed with particular reference to shock. Roughly, there were two main types, the slowly twisting type of volvulus and the quickly twisting type, the latter of which is highly dangerous when diagnosis is delayed.

The mortality rate was high (14 of 39 cases dying), due mainly to late diagnosis with consequent delay in hospitalization and treatment. However, there were only 2 deaths in the last 14 cases and one was not actually due to the volvulus.

The follow-up disclosed no recurrence of the volvulus in any instance.

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ADENOCARCINOMA OF THE KIDNEY

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IT is impossible to arrive at an accurate estimate of the behaviour of disease without an intensive survey of the obtainable information, and accuracy is not attained unless an adequate number of cases is assessed. In an attempt to demonstrate the behaviour of carcinoma of the kidney, and in particular the prognosis following nephrectomy, 100 consecutive cases of this disease are presented.

Historical Aspects

In 1861 Rokitansky, when describing renal tumours, stated that very often in carcinomata considerable haemorrhagic foci were present and large areas were undergoing fatty metamorphosis. Such a description is clearly applicable to the typical tumour of variegated appearance, known today as carcinoma of the renal parenchyma.

Twenty-two years later Thornton (1883) removed a renal tumour and described it as "alveolar sarcoma of the capsule of the kidney." Portion of this specimen was examined microscopically and the report was: "Both in the arrangement of the cells in columns, and also in the shape and general appearance of the cells themselves, the tissue greatly resembles the suprarenal capsule."

In the same year (1883) Grawitz also proposed the hypothesis of suprarenal origin and for the next ten years this was generally accepted. During this period Sudeck (1893) attacked the hypothesis, considering the tumours to be tubular adenomata of the kidney, but little note was taken of Sudeck's proposition and the Grawitz viewpoint was further strengthened by the confirmatory opinion of Lubarsch in 1894.

However, during this present century, beginning with a paper by Stoerk in 1908, there has been an accumulation of observations opposing the adrenal theory. Stoerk pointed out the profound macroscopic difference between a tumour of the adrenal gland and a tumour found beneath the capsule of the kidney, and he noted further that, though tumours of the adrenal were uncommon, tumours of the kidney were a frequent occurrence.

Glynn in 1911 stated that tumours of the adrenal were far more common in the female, but that tumours beneath the capsule of the kidney showed a striking preponderance in the male. He was also the first to point out that changes in sex characteristics occurred with adrenal tumours but not with renal tumours, and to date no hormonically active renal tumour has been described.

One of Grawitz's arguments in favour of his hypothesis was his belief of a common lipoid contained both in the suprarenal and also in the subcapsular renal lesions. That the lipoid substances in these two sites is far from identical has now been abundantly proven by biochemical analysis. The weight of evidence has thus shifted and the tumour, which has masqueraded under the titles of "Grawitz tumour," "hypernephroma," "sarcoma," "carcino-sarcoma," etc., has now been widely accepted as a single entity known as carcinoma of the renal parenchyma or adenocarcinoma of the kidney.

Such a view, however, is not yet universal. As recently as 1940 MacCallum in his "Textbook of Pathology" classified renal neoplasms of two main types:

(i) tumours of adrenal origin (hypernephromata);

(ii) true adenocarcinomata of the kidney —passing mention only being given to this group.

Material

This series comprises 100 consecutive cases of carcinoma of the kidney which have been compiled from the records of the Royal Melbourne Hospital, the Alfred Hospital, and Prince Henry's Hospital during the periods 1940-1941 and 1946-1953. A certain selection has been exercised in that no case was included unless the diagnosis had been proven

microscopically. A large number of cases with strong presumptive diagnoses were thus rejected.

CLINICAL ASPECTS

History

The renal triad of pain, haematuria and mass was present in only 25 cases.

The commonest single symptom was haematuria, this being present in 55 patients. In 17 instances the haematuria had been an isolated event before presentation at hospital, while 38 patients had experienced periodic episodes of blood in the urine.

Pain was nearly as frequent as haematuria, being a feature in 52 cases. The majority of the patients, that is 25 in number, complained of severe colicky pain and in only 17 cases was the pain regarded as not colicky in type. Pain was closely related to the presence of blood in the urine and in only 4 cases was this present without haematuria.

Loss of weight was a common symptom and 41 patients in this series had noted a loss in weight of one stone or more.

In 9 instances the presenting symptom was due to a secondary manifestation. In four of this group there were bony metastases and in 2 cerebral metastases. The presenting feature in one case was a lump in the neck, while the final case of this group presented with dysponea due to pleural effusion.

There thus remains the large group of patients (37 in number) who were admitted to hospital with no history relating to the renal tract.

The examination

Clinical examination of the cases showed the following features:

(i) Mass. A palpable mass was demonstrable in 37 cases.

(ii) Urine microscopy. Red blood corpuscles were detected in the urine in 37 cases, macroscopically or microscopically. In 3 instances, blood in the urine was demonstrated microscopically when no history of haematuria had been obtained. This is of interest as renal tumour is often unsuspected and in this series there was no record of urine microscopy in 23 patients.

(iii) Haemoglobin. This was recorded in 51 cases and in 12 cases this was 60 per cent. or less. Of these 12 cases, 6 gave no history of blood loss.

(iv) Temperature. The temperature on admission to hospital was 99°F or more in 32 of the 100 patients.

Diagnosis

Where the patient gave a history referable to the renal tract the diagnosis of renal carcinoma was usually established clinically. Clinical misdiagnosis did occur, occasionally, hydronephrosis being the diagnosis in 2 cases, tuberculosis in two others. These impressions were corrected at operation.

Interest lies mainly in the establishment of a diagnosis in the 37 patients with no history of renal disturbance. Here the diagnosis was largely dependent on the presence or absence of a palpable abdominal mass.

Group A—Cases with an abdominal mass—16 patients:

(i) carcinoma of the kidney was correctly diagnosed in 8 patients. In one case of this group, the diagnosis of "pyrexia of unknown origin" was excluded only after many weeks, and when the plethora of investigations had finally included an intravenous pyelogram;

(ii) the diagnosis of abdominal carcinoma, primary site unknown, was made in 5 cases;*

(iii) malignancy was unsuspected in 3 cases.

Group B — Cases where no mass was palpable—21 patients:

(i) malignancy was not suspected in 11 patients;

(ii) the clinical diagnosis of "malignancy, primary site unknown" was made in 6 cases;*

(iii) four cases remain in which the diagnosis of renal malignancy was made, and in this group the diagnosis was established with some difficulty. Thus one patient underwent excision

*Patients not fit to withstand full investigation.

of a varicocoele, the carcinoma remaining unsuspected until three months later when a swelling appeared in the loin.

Pyrexia of unknown origin was excluded again in one case by the ultimate use of pyelography. The two remaining patients underwent operation with no clinical suspicion of renal malignancy, one being diagnosed at craniotomy for a supposed primary cerebral tumour. The last of these cases illustrates the need of pathological proof of the diagnosis. This patient was considered to have amoebiasis and had in fact "responded" to two courses of emetine therapy. Not until laparotomy to drain an "amoebic abscess" was undertaken, was the true picture obtained.

Thus, of the 37 patients with no renal history, 14 finally reached autopsy with no prior clinical suspicion of malignancy.

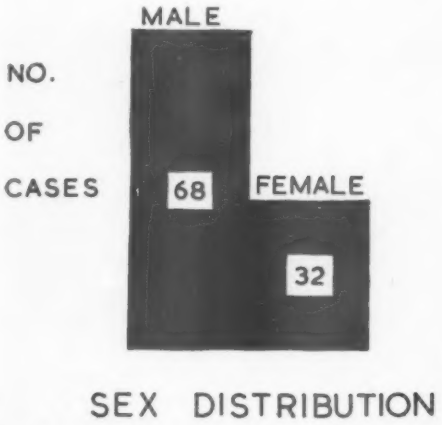


FIG. I. Graphic illustration of the sex incidence of carcinoma of the kidney.

MACROSCOPIC PATHOLOGY

Sex incidence

In agreement with almost all recorded series, the greater number of cases occurred in the male sex. In this series 32 of the patients were females, 68 being males (Fig. I).

Side

Fifty-two cases occurred on the right side, 48 on the left side. Although some authors state that tumours of the right kidney are twice as common as those of the left (Herbut, 1952), this finding of almost equal occurrence on the two sides is again in accord with most recorded series.

Age

The average age at death was 58 years, the oldest patient being 86 years. Below the age of 40 the condition is uncommon, 3 cases only being found in this series, the youngest of whom was 36 years. Willis (1953) reports the tumour as being rare in the young, the youngest in his series being 29 years. Nicholls (1936) has reported a typical papillary adenocarcinoma in a child of 22 months (Fig. II).

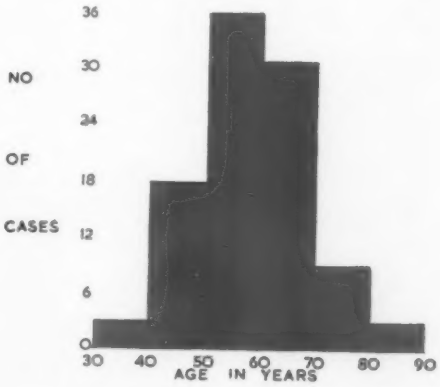


FIG. II. Nomogram to illustrate the age incidence of the tumour; the maximum incidence being in the fifth and sixth decades.

General description

The small tumour was always found to be cortical in position and commonly situated towards either the upper or lower pole. The size of the tumour varied greatly, the largest tumour in the series being 26 cm. in diameter. The average diameter of the operative specimen was 5-10 cm. At autopsy it was quite common to find a small tumour of 1-3 cm. diameter, but a tumour of this size was seldom seen at operation. Typically the neoplasm was solitary and circumscribed. The outstanding feature on gross section was the variegated appearance, this being due to the

division of the tumour into lobules by fibrous septa and the presence of tissue of differing nature within each lobule.

FIG. III. Graph to represent the survival in months of the 43 patients who had had a nephrectomy, and who died since operation. The gradient of the curve shows that over 50 per cent. of these patients died within twelve months of operation.

tumour thus loses its variegated appearance, and becomes off-white to yellow in colour. Some evidence of haemorrhage, however, is usually found at the periphery of the necrotic tissue. Another feature may be superimposed, for cystic changes are often associated with the necrosis.

MICROSCOPIC PATHOLOGY

All sections examined had been stained with haematoxylin and eosin.

The cellular arrangement

The better differentiated the tumour, the more regular the arrangement of cells which may then present a regular adenomatous or papillary pattern. The more anaplastic tumour departs from this uniformity, the cells being disposed haphazardly or occasionally in sheets.

The stroma

Generally the connective tissue exists as thin strands running throughout the tumour but presents wide variations, and in the anaplastic tumour the connective tissue itself may assume the appearance of malignancy.

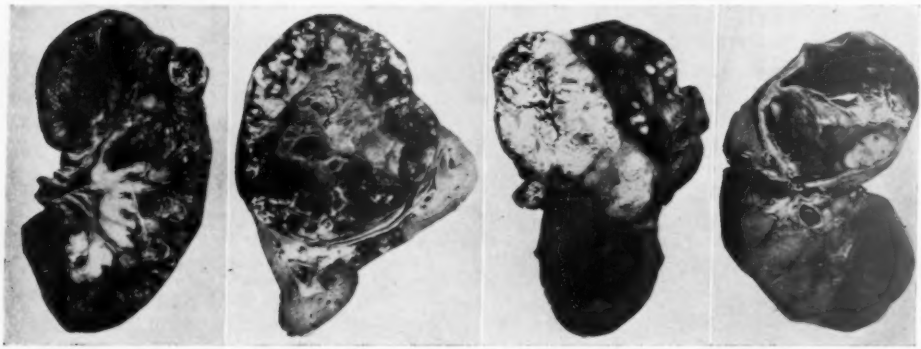


Fig. IV Fig. V Fig. VI Fig. VII

FIG. IV. Photograph of the early lesion, situated in the cortex and giving no symptoms.
FIG. V. Photograph illustrating the typical variegated appearance of the well-developed lesion; it demonstrates the haemorrhagic nature of the lesion.
FIG. VI. Photograph showing the changed macroscopic appearance, when the tumour becomes necrotic.
FIG. VII. Photograph demonstrating that cystic changes occur in association with necrosis.

Characteristically the tumour is extremely haemorrhagic and on close macroscopical inspection, its papillary structure can often be visualized. Necrosis is a common development in this tumour, and when a dominant feature, haemorrhage is not prominent except at the periphery of the necrotic areas. The

The nuclear pattern

The nucleus is oval—almost round—and subject to great variation in size in different areas of the same tumour, and even in the one microscopic field. It contains usually a single deeply-staining nucleolus which is

eccentrically placed, the rest of the nucleoplasm being finely granular. Despite the undoubted malignancy of the tumour, mitotic figures are not a feature, though multiple nucleoli are not uncommon.

Less commonly the nucleus may appear as a uniformly staining, deeply basiphilic mass.

The cytoplasm

The cytoplasm may stain with eosin, and this staining may be either uniform or irregular. In the latter case the cytoplasm assumes a granular appearance, but more commonly the cytoplasm does not stain at all and appears "clear" or may only stain in strands. All varieties exist in the same tumour.

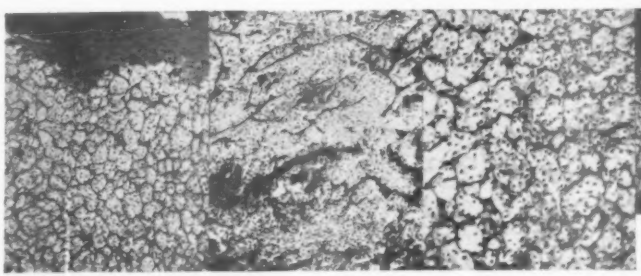
death of the patient. The conclusion drawn was that the histological picture of highly differentiated tissue, usually associated in other kinds of growth with slow growth of a lesion, did not necessarily warrant a cheerful prognosis in this particular tumour.

AUTOPSY FINDINGS

Autopsy was conducted in 48 cases. Of these metastases were present in 35 cases and in 13 instances, no metastasis was demonstrable.

The local lesion

In this group the figures refer to both operative and post-mortem specimens.



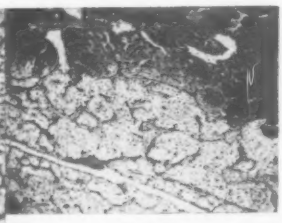


Fig. XI

Fig. VIII Fig. IX Fig. X

FIG. VIII. Photomicrograph showing a "clear cell" carcinoma arranged in a regular but non-papillary pattern. (x 150)

FIG. IX. Photomicrograph again shows the cellular pattern which in this case is more of papillary arrangement. (x 35)

FIG. X. Photomicrograph in which the cytoplasm is "clear" in some areas, finely granular in others. (x 150)

FIG. XI. Photomicrograph illustrating that the staining of the cytoplasm is subject to great variation, as is the cellular arrangement. In the upper portion the cells are disposed in sheets and the cytoplasm stains deeply. In the lower part the adenomatous pattern is preserved and the cytoplasm is again "clear."

Relation to prognosis

In this series no constant relation between histological picture and survival time could be made. In one case the differentiation of the tumour and the well-developed tubular pattern was so definite that the pathological diagnosis of benign adenoma was made. Two years later this patient died with multiple metastases, all of which now presented a histological picture of extreme anaplasia.

Thus while the histological picture may be diverse, varying from the seemingly benign to the obviously malignant, it was found that many of the apparently well-differentiated tumours gave rise to early metastases and

Extension of tumour tissue beyond the capsule was noted in 25 cases.

In 28 cases, it was noted that tumour tissue encroached into the renal pelvis. All of these patients had noted haematuria as a symptom. The renal vein was invaded in 27 cases and tumour tissue had further extended into the inferior vena cava in 8 of these.

It should be noted that these figures are probably a very conservative estimate as, in over one-third of cases, there was no record of careful examination of the kidney, nor was there record of extension beyond the renal parenchyma.

Metastases

The sites of macroscopic metastases, all of which were later confirmed by microscopy, were as follow:

Lungs	23
Liver	18
Retroperitoneal	13
Bones	11
Lymph Nodes	9
Other Kidney	4
Brain	3
Heart	3
Spleen	2
Ovary	2

With a series of only 100 cases and inadequate observations in some instances, it is difficult to draw accurate conclusions, but the following correlations may be made:

(i) haematuria occurred in all cases in which tumour tissue could be demonstrated in the renal pelvis;

(ii) extension beyond the capsule was a feature in 25 instances. In only 2 of these patients was it found impossible to remove the kidney at operation;

(iii) renal vein involvement occurred in 27 patients, extending into the vena cava in 8. All but one of this group complained of severe weight loss.

TABLE 1

SHOWING POST-NEPHRECTOMY AUTOPSY

Case No.	Survival in months	Site of metastases
1	Nil	Nil
2	Nil	Pancreas, spleen, liver, other kidney, heart, lungs, skull, femur
3	Less than 1	Lungs
4	Less than 1	Other kidney, retroperitoneal
5	1	Suprarenal
6	1	Suprarenal, retroperitoneal, lungs, heart, lymph nodes
7	3	Liver, spleen, retroperitoneal
8	12	Suprarenal, peritoneum, omentum, pleura, liver
9	26	Retroperitoneal, liver, ovary, lungs, lymph nodes
10	34	Retroperitoneal, liver, pancreas, lungs
11	51	Lungs, bones, lymph nodes

Willis (1953) states that metastases are present in the skeleton in nearly one-half of fatal cases, while the brain contains metastases in about one-quarter of such cases. The figures in this series are much lower than the ones which Willis quotes particularly with regard to those found within the cranium. This discrepancy can be explained by the fact that in this series, in over one-third of cases, the skull was not opened at post-mortem examination.

Autopsy was obtained in 17 of this group and in 14 metastases were present.

Post-nephrectomy autopsy

Nephrectomy was performed on 61 occasions. Of these 61 patients autopsy records are available for 11 of these patients who eventually died in hospital. In all instances the growth in the kidney was considered clinically to be the only lesion present. It is

therefore of some interest to note that, in the 11 cases where a complete record from diagnosis to death is available, there was only one case in which no macroscopic metastasis could be found. This isolated case occurred in a patient dying on the seventh post-operative day from pulmonary embolism.

Treatment

Nephrectomy was attempted on 63 occasions and was abandoned twice, it being found impossible to remove the kidney. Radiotherapy as the sole treatment was employed twice for 2 patients with known metastases.

No active treatment was given to the remaining 35 patients. Of these, renal malignancy was unsuspected in 16, the remaining 19 being too ill to withstand adequate investigation, let alone active treatment.

Prognosis

In an attempt to establish accurate survival times a search of the records of the public hospitals, the cancer registry and, in some cases, of the electoral rolls has been conducted.

Forty-three of the 61 patients are known to be dead. Eight patients are known to be alive. The survival period of these 8 patients is as follows:

Case 1 ..... 58 months
Case 2 ..... 40 months
Case 3 ..... 28 months
Case 4 ..... 28 months
Case 5 ..... 25 months
Case 6 ..... 20 months
Case 7 ..... 15 months
Case 8 ..... 4 months

TABLE 2

SHOWING POSSIBLE SURVIVAL OF THE 10 PATIENTS WITH WHOM CONTACT HAS BEEN LOST

When last contacted	Survival in months if still alive
At operation	37*
At operation	36
At operation	28
At operation	24*
At operation	24
At operation	19
At operation	18
4 months after operation	20
15 months after operation	100*
48 months after operation	61

*Not on electoral roll.

Operative mortality

Sixty-one patients underwent nephrectomy and within two weeks of operation, 6 patients were dead. Pulmonary embolism, bronchopneumonia and multiple metastases were the causes of death in 3 patients. In one case no convincing cause of death was established and in 2 cases autopsy was not obtained.

In the other 10 cases it has not been established with certainty whether the patient is alive or dead.

The known facts relating to survival times of these 10 cases is given in Table 2.

The hospitals were in receipt of information of the post-operative course of three of these patients for varying periods. As can

be seen, they have not been heard of for many months and it is most likely that they are no longer living.

No conclusions can be drawn of the 7 patients for whom there is no post-operative follow at all, but it is perhaps significant that at least two are no longer on the electoral rolls.

As previously stated 43 of the 61 patients who have undergone nephrectomy are now dead. Their survival in months following nephrectomy is best expressed graphically (Fig. III).

The largest series was published by Riches and his associates in 1951. These figures are referable to a total of 685 patients who have been followed since operation. Survival times for this group are as follows:

3 year survival .... 44 per cent.
5 year survival .... 30 per cent.
10 year survival .... 17 per cent.

These figures throw a far from cheerful light on the prognosis of carcinoma of the kidney and the malignancy of the tumour is further emphasized by the observation that in this series there is as yet (at November, 1954) not one authenticated five-year survival.

TABLE 3

SHOWING SURVIVAL TIMES IN RELATION TO DURATION OF HISTORY

History	Survival in months	History	Survival in months
1 month or less	28 still alive	4-6 months	4
	24		3 (2 cases)
	20 still alive		2
	15		1 (2 cases)
	11		Less than 1 (4 cases)
	6		
	5	7-9 months	25
	1		24 not traced since
	Not traced (2 cases)		8
2-3 months	43		1
	40 still alive		Less than 1
	40	10-12 months	44
	28 still alive		15 not traced since
	25		6
	15 still alive		3
	14		Not traced
	11		
	8	Over 12 months	
	1	18 months	13
	Less than 1 (2 cases)		Less than 1
4-6 months	58 still alive	21 months	48 not traced since
	52		
	51	22 months	3
	Not traced	24 months	24 not traced since
	24		9
	23		
	20	36 months	9
	18		
	17 (3 cases)	60 months	30

Figures obtainable from other sources are of interest. Priestley in 1939 published the following figures:

3 year survival .... 47 per cent.
5 year survival .... 38 per cent.
10 year survival .... 27 per cent.

Factors influencing prognosis

In common with other series (Herbut, 1952; Riches, 1951; Priestley, 1939) it was noted that the prognosis was adversely affected by the involvement of the renal vein by tumour tissue.

As it is often stated that the surgical cure of carcinoma depends on the early detection and early surgical ablation of the lesion, particular interest attaches to the relation between the length of the history and the survival of the patient. In this series it was found that expectancy of survival was not increased if surgery was undertaken early, and in support of this statement, Table 3 is appended to show the history-survival correlation. If any conclusions can be drawn, it would appear that the patient with the long history of renal disturbance had a better prognosis than that with the short history.

SUMMARY

One hundred cases of adenocarcinomata of the kidney are presented, all of which have been proven microscopically. Because of lack of histological proof, many probable cases have been excluded.

The commonest symptom is haematuria and this was present in all cases when the tumour had involved the renal pelvis.

Invasion of the renal vein, loss of weight and metastases are common companions.

It would appear that metastases are often present before nephrectomy is performed for an apparent local clinical lesion.

The prognosis of the patient with this lesion is not improved by early surgical ablation.

The malignancy of renal carcinoma is emphasized.

ACKNOWLEDGEMENTS

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MANAGEMENT OF CORROSIVE BURNS OF THE OESOPHAGUS

By JOHN BORRIE

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THE key to successful management of corrosive burns of the oesophagus lies in clearly appreciating the progress of its pathology from burn to stricture formation.

PATHOLOGY

The severity of the lesion depends upon the corrosive agent swallowed, its chemical nature, concentration, quantity and duration of contact with the oesophageal tissues (Daly, 1954).

Alkalis, especially caustic soda and ammonia, are the commonest substances taken, but sulphuric and nitric acids, lysol and iodine have all been encountered.

Whereas acids produce a coagulation necrosis which limits their action to the superficial layers of the oesophagus, alkalis, producing a liquifying necrosis, penetrate to the deepest layers and excite an intense inflammatory reaction. Lysol produces a superficial oesophagitis only, and no stricture results.

The severest burns occur in the middle and lower thirds of the oesophagus.

There are three distinct phases with a severe burn; the acute phase of local reaction to the burn, the intermediate phase of ulceration and healing by granulation tissue, and the final phase of fibrosis and stricture formation. These correspond to the clinical phases.

(1) The acute phase, which lasts up to two weeks, commences with superficial necrosis and mucosal sloughing, followed by inflammatory oedema of all layers of the oesophagus.

(2) In the intermediate phase, the slough separates, and passes down the alimentary canal leaving behind angry red granulations covering a tubular ulcer.

(3) In the final phase there is gradual replacement by fibrous tissue which contracts to stricture formation. The severity of the final stricture is closely related to the adequacy of early treatment. The less the treatment, the greater the chance and degree of stricture formation.

CLINICAL FEATURES

Clinically cases fall into three groups:

(a) those with mild burns;

(b) those with severe burns and no other complication, and

(c) those where complications accompany severe burns.

The last are usually fatal. This paper deals particularly with the second group.

In the acute phase with associated burns of lips, mouth, tongue and pharynx, the patient is in great distress, and has retrosternal pain. There is also dysphagia, incomplete or complete, when signs of dehydration and even acute starvation are added.

In the intermediate phase of two to four weeks duration the dysphagia improves, soft food and liquids are readily taken, and there is an unreal feeling that all is well.

In the final phase, progressive dysphagia returns, until, when the stricture is finally established, everything that is swallowed is regurgitated.

DIAGNOSIS

Whenever a patient presents with a history of having swallowed a corrosive substance, it is important to establish:

(a) that an oesophageal burn actually exists;

(b) its severity, and

(c) stage to which it has progressed.

This can only be accurately determined by oesophagoscopy, nor should this step be delayed in the absence of an obvious oral burn.

Fluoroscopy is more important later, in determining the site and nature of any subsequent stricture of the gullet.

THE MANAGEMENT OF THE ACUTE AND INTERMEDIATE PHASES

The immediate problems are to:

(1) Neutralize the chemical agent;

(2) relieve the symptoms;

(3) maintain nutrition;

(4) counteract the infection with chemotherapy, and

(5) counteract possible stricture formation.

Unless instituted immediately following the burn, it is doubtful if neutralization has any effect on the subsequent course of events. Emollients such as olive oil are certainly soothing.

Krey (1952) in an experimental study on rabbits, has shown that a normal solution of sodium hydroxide in contact with oesophageal mucosa for 10 seconds necrosed mucosa and submucosa to involve some inner longitudinal muscle fibres, 3N solution necrosed to the circular muscle layer, and 5N to the outer longitudinal muscle. A 7N solution penetrated through the entire oesophageal wall and into the peri-oesophageal tissue.

There are two methods of counteracting probable stricture formation:

(a) by attempting to limit granulation tissue formation;

(b) by maintaining the oesophageal lumen.

The former is in part controlled by chemotherapy which limits the intense granulation reaction to severe infection, and is possibly also affected by ACTH.

More important is to preserve an oesophageal lumen.

The first line of approach is daily oesophageal bouginage, which should be initiated the first day. If a wide-bore stomach tube is used, then high protein fluid diet can be run down the tube. It is important to persevere for at least two months, and not be misled by early return of apparent ease in swallowing.

If the discomfort of this treatment proves too great, the speed of stricture formation too rapid — or if the lesion is seen for the first time in the intermediate phase, a wide-bore (40 French) stomach tube placed within the oesophageal lumen is required. This becomes both a food passage and an internal oesophageal splint to preserve oesophageal lumen during the stage of cicatrization. In order to avoid pulmonary complications the upper end of the tube is brought out on to the neck through a cervical oesophagostomy as described by Leegaard in 1945.

The complexities of management of the acute and intermediate phases are well shown in the following case report:

L.T., a soldier aged 26 years, who had returned from Korea in August, 1952, was treated for three months in Seacliff Mental Hospital, Otago, for acute schizophrenia. On 30th December, during Christmas leave, he attempted suicide by swallowing caustic soda, and at midday on 31st December, 24 hours afterwards, he was admitted to Dunedin Hospital.

He complained of great pain in his pharynx and behind the sternum, and continually spat all saliva into a large basin. Clinically he had burns of his lips, and tongue, and had a sloughing pharyngeal mucosa. Salivary secretion was lessened with atropine, gr. 1/100 four hourly. Oesophagoscopy under general anaesthesia showed the burn to have involved the entire oesophagus which had a white sloughing mucosa. As a Levine tube could not be made to enter the stomach, intravenous fluids were given.

On the second day after the burn, 1st January, 1953, under local sedation and local anaesthesia in his mouth, a stout stomach tube (30F) was successfully passed into the stomach, and high caloric fluid diet mixture given. This manoeuvre was repeated twice on the third and fourth days.

On the fifth day, as he could swallow a fluid diet normally, all artificial feeding and passage of wide-bore stomach tubes was stopped.

On the tenth day further oesophagoscopy showed that the burn had progressed from the acute phase to the intermediae phase of healing with eventual fibrosis. From 17 cms. onwards the oesophagus was lined by red granulation tissue. Already the upper oesophagus tightly gripped the oesophagoscope. One wondered at that time if an indwelling tube should be inserted.

On the seventeenth day, as complete dysphagia had returned, further oesophagoscopy was performed. Dilators, however, could not be passed into the stomach.

It was obvious that, if tight stricture were to be avoided, an internal oesophageal splint was required, and that the only way of getting it through the oesophagus was from below. The left transthoracic route was chosen in preference to the abdominal route as giving greater view of the oesophagus, especially as the possibility of oesophageal perforation at the previous oesophagoscopy had been raised.

At operation, the oesophagus, which was greatly oedematous, was freed from the mediastinum. It was not possible to detect any site of perforation. A

catheter sling was placed round the oesophagus as a retractor, the diaphragm was radially incised and the fundus of the stomach opened. A wide-bore stomach tube was then successfully passed up through the cardia and guided through the whole length of the oesophagus to the mouth. A second stomach tube was tied to this in reverse, and, by pulling on the first, the second stomach tube was guided in from the mouth and into the stomach. The gastrotomy wound was closed with chromic catgut, reinforced with a layer of interrupted silk sutures and omentum, and the diaphragm and chest wall closed in layers with interrupted silk sutures.

At the end of the operation left cervical oesophagostomy was performed and the stomach tube brought out of the neck just below the cricopharyngeal sphincter. The cervical wound was then packed with vaseline gauze (Fig. I).

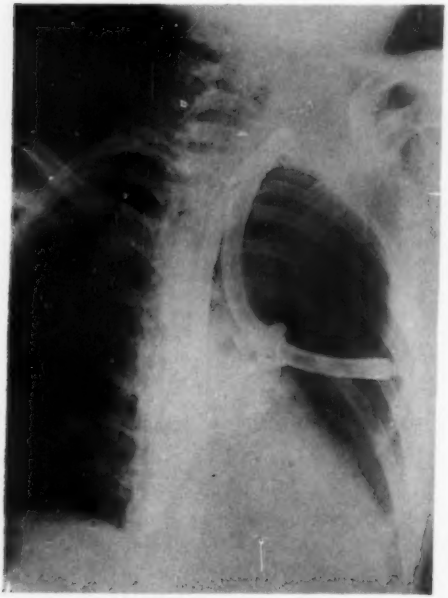

FIG. I. Stomach tube, entering through left cervical oesophagostomy acts as internal oesophageal splint and food passage.

The wounds rapidly healed. The patient had this internal oesophageal splint in position for the next eight weeks, when it was removed and daily dilatation commenced with large gum elastic bougies. Under this regime progress was well maintained and serious stricture formation thus prevented (Fig. II). He continued this daily oesophageal bouginage for the next year, and gradually extended the intervals between passing of the bougie to weekly intervals. Eighteen months later he was swallowing normally.

FIG. II. Barium swallow five and a half months after caustic burn.

Comment: In this case, had one persisted for two months with twice daily passage of a wide-bore stomach tube or gum elastic bougie, it is likely that the onset of stricture by the

seventeenth day after the burn could have been avoided. As it was, ten days treatment were lost in the belief that all would be well. When the stricture finally declared itself it was impassable from above. The transthoracic approach showed that there was no oesophageal perforation after attempted bouginage, and greatly facilitated the passage of the stomach tube up the oesophagus. Convalescence was thereafter uneventful.

MANAGEMENT OF THE ESTABLISHED STRICTURE

With an established oesophageal stricture, fluoroscopy and oesophagoscopy are again required as the first step in determining its site and nature. As carcinomata have been known to develop on existing chemical strictures, biopsy is essential.

The aims of treatment are:

(a) To dilate the stricture if possible and and keep the oesophagus patent.

(b) If this proves unsuccessful, to restore oesophageal continuity by surgical means.

(a) Dilatation of stricture

There are three methods of dilatation:

(1) From above using Jackson bougies.

(2) From above with bougies threaded over a swallowed string.

(3) From below—retrograde dilatation.

For satisfactory dilatation of a difficult stricture, good general anaesthesia with endotracheal intubation is essential. At first fine ureteric catheters may have to be threaded through the tight stricture and gradually replaced by bougies. After several twice weekly treatments, it is possible to get size 26 French dilators through.

If a thread has previously been able to pass the stricture, then it acts as a guide for hollow dilators, and thereby lessens the risk of perforation of the oesophagus.

If the stricture cannot be passed from above, then gastrostomy is required. Once the oesophagus has been rested from previous manipulations retrograde dilatation should be tried. This can be facilitated if a thread has previously been swallowed and has entered the stomach when Tucker bougies can be passed up in series. Howard (1954) has found this method invaluable. Once the stricture is passed from below, dilatation is continued from above. At a later date self-bouginage is taught.

For self-bouginage it is important to use only French gum elastic bougies in good condition. Force should never be used, nor roughness, and the patient should not experience pain. The bougie must be passed each day and left *in situ* so long as the patient can tolerate it, preferably for twenty minutes.

Diet: At first the patient can take only a fluid diet, but later, as the stricture dilates up, purée is added. Finally the patient should be encouraged to eat as much well-chewed solid food as is possible. In Grey Turner's experience, food itself, in time, proved to be a useful oesophageal dilatator.

(b) Surgical treatment

Surgical treatment is indicated where:

(1) The oesophageal stricture proves unyielding to conservative measures.

(2) It continues to recur with increasing frequency despite repeated dilatation.

(3) Where it is economically unsound for young patients with domestic responsibilities to be repeatedly attending hospital.

Type of operation: The ante-thoracic oesophageal reconstruction, described by Yudin (1944) on 80 patients, must nowadays be considered largely of historic interest only. It is superseded by the far greater advantages of endo-thoracic oesophageal reconstruction.

There are two types of operation:

(i) Where the stricture is less than 3 cm. long, local resection can be considered with end-to-end oesophageal reconstruction.

(ii) Where the lesion is longer, the treatment of choice is oesophago-gastrostomy without excising the stricture (Rapant and Hromada, 1950).

(a) Partial oesophagostomy and end-to-end oesophageal reconstruction

This operation, of which I have no personal experience, was first described by Gross in 1948, and performed on a child 17 months old with a short oesophageal stricture in the lower third of the oesophagus. Pre-operative gastrostomy had been required.

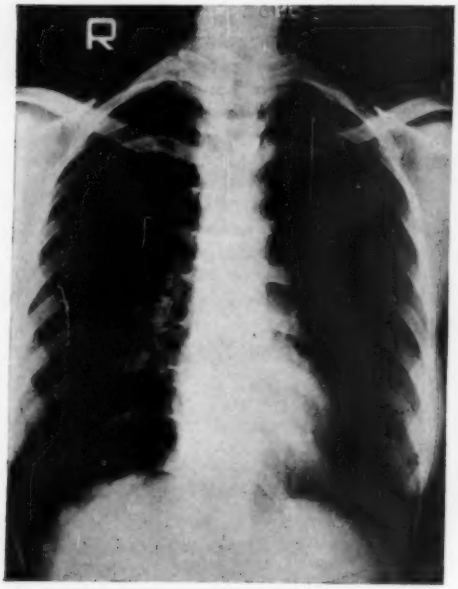

FIG. III. Blind oesophageal stump at clavicular level twelve years after caustic burn.

Gross advised that where the exact length of the stricture is known, and oesophageal reconstruction therefore known to be feasible, then the right-sided approach should be favoured. Where, on the other hand it is difficult to predict if oesophageal anastomosis is feasible, and where it could be waved in favour of oesophago-gastric anastomosis, then the left-sided approach should be preferred. The oesophagus is raised from its bed, gently mobilized, the upper limit of the stricture defined with the aid of a stiff catheter passed through the mouth by the anaesthetist. The oesophagus is divided at that level. The lower limit of the stricture is reached by probing, and the oesophagus again divided at that level. Oesophageal re-suture is effected with a two layer anastomosis using 4-0 Deknatel suture. The chest is closed over water-seal drainage which is maintained for four days. In these cases, if gastrostomy has not already been made, it should be carried out to ease the problems of feeding and to give the anastomosis the best chance of healing.

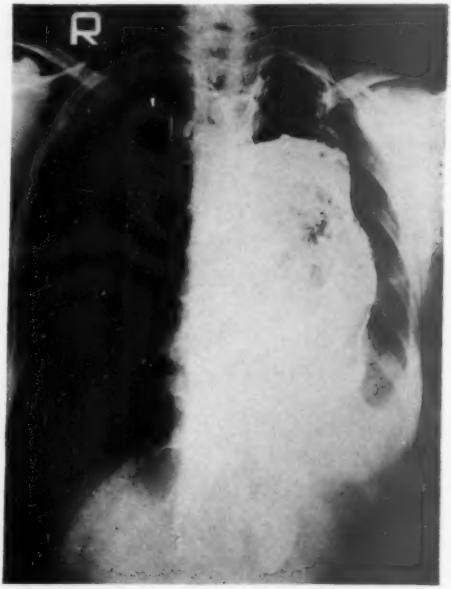

FIG. IV. After oesophago-gastrostomy without excision of stricture.

(b) Oesophago-gastrostomy

In the presence of a long intractable stricture of the oesophagus, high intra-thoracic oesophago-gastrostomy is required. The important features are shown in the following case report:

Mrs T.R.T., a Maori woman aged 24 years, was seen by me and admitted to Green Lane Hospital, Auckland, on 16th June, 1952. Twelve years before when she was 12 years old, a friend gave her a "lemon squash" drink, which proved to be caustic soda. She was then living forty miles from the nearest medical aid, nor was it possible to reach hospital for a fortnight because of flooded, unbridged rivers. When admitted to hospital, gastrostomy had been promptly performed as a life-saving measure. She continued with her gastrostomy all these years, she had married and had borne one child, but had never been able to eat anything by mouth. To say the least, her life was socially embarrassing.

Barium studies and oesophagoscopy showed a dilated oesophageal remnant in the superior mediastinal and normal oesophageal mucosa to the level of 22 cm. from the upper incisor teeth. Beyond that was an impassable stricture (Fig. III).

On 2nd July, 1952, oesophago-gastrostomy was performed by Mr. Douglas Robb. The left chest was opened through the bed of the resected left 9th rib, the diaphragm was radially incised, the stomach detached from the ventral abdominal wall and the gastrotomy opening closed. The proximal end of the stomach was mobilized, but the pyloric blood supply preserved. The stomach was then taken into the left pleural cavity and the diaphragm closed around it. The chest wall was closed; and, at the second step, with the patient now lying on her back, the medial half of the left clavicle and first rib were resected. Cervical oesophagus was isolated and oesophago-gastrostomy performed between the upper pouch and the fundus of the stomach. The patient made an uneventful recovery from her operation (Figs. IV and V).

FIG. V. The site of anastomosis.

Comment: In this case the segment comprising middle and lower thirds of oesophagus, which had been completely occluded for 12 years, was completely isolated by the operation. It was felt that the very real hazard of excising this segment was unjustified, indeed not necessary, and that any residual retention cyst formation of the oesophageal remnant most unlikely. Certainly two and a half years after operation none has appeared. This had been the clinical experience of Rapant, Hromada, and Mekie (1954), and had also been confirmed experimentally by Denton and Bradshaw (1952).

RESULTS

Though small in number, the results of surgical resection of chemical stricture are encouraging. Gross in 1950 reported 4 successes from local resection and end-to-end anastomosis of the oesophagus.

In 1946 Sweet published 3 successes following sub-total oesophagectomy. Of 4 such operations, Puestow and Chess (1948) reported 3 recoveries, and 1 death associated with hydropneumothorax, collapsed left lung and bronchopneumonia in the right lung. One of the 3 successes developed a temporary leak from the anastomosis which responded satisfactorily to drainage.

In 2 patients with stricture involving the middle half of the oesophagus, Rapant and Hromada (1950) performed oesophago-gastrostomy without resection of the stricture through a right thoracotomy. In 2 other patients, with stenosis of the entire thoracic oesophagus, the mobilized stomach was brought up into the left chest and anastomosed to the cervical oesophagus through a second incision. The left clavicle was divided and the left first and second ribs resected to facilitate anastomosis. All 4 patients are well and relieved of symptoms.

Mekie (1954) was able to perform oesophago-jejunostomy in 7 cases, of whom 5 survived with satisfactory results. In none did he attempt resection of the oesophageal remnant, nor had any developed an oesophageal cyst.

THE LATENT STRICTURE

Many years after a burn, the patient may develop acute dysphagia, because, in a moment of forgetfulness, he has swallowed partly chewed food which has effectively blocked an old, incompletely dilated stricture.

Case report

K.C., a youth aged 21 years, was admitted as an emergency to the Dunedin Hospital on 20th December, 1953. At the age of three years he had swallowed two teaspoonfuls of caustic soda and since then had an oesophageal stricture which had yielded to dilatation (Fig. VI). Throughout his youth he

frequently had episodes of complete dysphagia from swallowing large pieces of unchewed food; but since the age of twelve years, he had had only three such attacks, the last occurring on Christmas Day, 1952.

Oesophagoscopy showed that the stricture at the lower end of his oesophagus was tightly packed with partially masticated meat. This was removed with immediate relief. Examination of the X-ray films showed that as a result of his previous caustic burn he had a long narrow oesophageal stricture (Fig. VII). As it was felt that this could be further dilated, he was instructed in the art of self-bouginage, since when there have been no further episodes of dysphagia.

FIG. VI. Stricture in 1937. Patient aged 5 years.

In conclusion, it is stressed that:

(1) Chemical strictures, especially in children, are preventable by strictest care with caustic in the home.

(2) Although the immediate problem in the acute phase is to neutralize the chemical substance, thus possibly lessening the severity of the burn and stricture formation, in the time required to find the antidote, the burn will have wrought its damage.

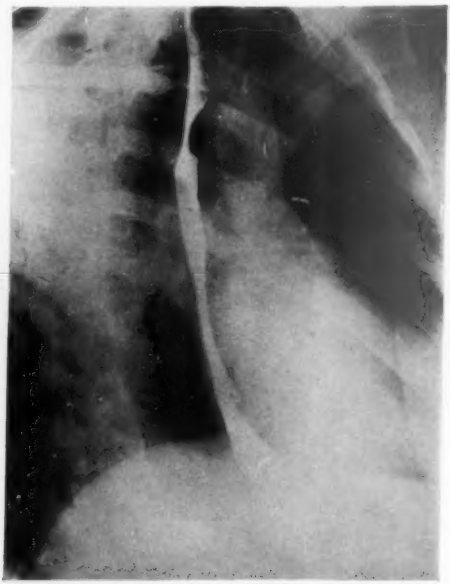

FIG. VII. Stricture in 1953 after temporary blocking by partly chewed meat.

(3) In the intermediate phase of healing, in order to forestall stricture formation in severe cases twice daily bouginage is necessary. If the discomfort of this treatment proves too great, the speed of stricture formation too rapid, or if the lesion is seen for the first time in the intermediate phase, then a wide bore stomach tube should be inserted into the stomach through a cervical oesophagostomy. It thus acts as both an internal oesophageal splint and as a pathway for feeding.

(4) Many established strictures will respond to careful dilatation with bougies.

(5) The patient with a dilated stricture must always masticate carefully to avoid temporary episodes of complete dysphagia from food blocking the narrowed oesophageal lumen.

(6) If the stricture proves unyielding it must be treated surgically:

(a) If small, less than 3 cms. long, and accessable by local excision and end-to-end oesophageal anastomosis;

(b) if longer, by oesophago-gastric anastomosis above the stricture level.

Surgical reconstruction not only restores normal swallowing, it restores a normal way of life.

ACKNOWLEDGEMENTS

I am grateful for the co-operation of Dr. Blake Palmer, Medical Superintendent of Seacliff Mental Hospital with Case 1, and Mr. Douglas Robb, Senior Thoracic Surgeon, Green Lane Hospital, Auckland, with Case 2. I also gratefully acknowledge photographic help from Miss Marshall, Department of Photography, Dunedin Hospital.

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FIBROMA OF VISCERAL PLEURA

By John Barnett

Alfred Hospital, Melbourne

WITH the recent increase in the number of people presenting themselves for chest X-ray, especially by the mass survey units, there has been an associated increase in the observed incidence of both benign and malignant intrathoracic tumours. Gould (1940) found an incidence of approximately 1% non-tuberculous lesions in a mass chest X-ray survey of over 400,000 persons and Rubinstein in a recent survey in Sydney designed primarily for the detection of tuberculosis, found 900 non-tuberculous lesions in 190,000 persons. Despite this overall increase however, the rarer benign tumours, often completely asymptomatic, are still not commonly seen even in the thoracic surgical units of large hospitals, and in this category can be included the true intrathoracic fibromas. Blades, in 1941, in a review of all reported intrathoracic tumours could find only 32 cases of fibroma, and many of these he considered to be fibrosarcomas or neurofibromas rather than true benign fibromas. Since his review there have been several isolated reports of cases but until recently all reports have been of single cases, so that they must be regarded as rare tumours. It is the purpose of this paper to present a further five cases, four of which were discovered by mass X-ray survey, and removed at thoracotomy over the last twelve months.

Clinical Features

The first four cases, 2 males and 2 females varying in age from 32 to 53 years, presented for examination after an abnormal lung shadow had been found at mass survey. Two of these patients reported that a mass survey film 2 years previously had been regarded as normal, but unfortunately these films are not available for re-examination in the light of the subsequent findings. One patient had suffered from asthma for the last five years. Otherwise they were asymptomatic and physical examination was completely negative.

The fifth case was a female, aged 55 years, who had reported to her doctor with an attack of upper abdominal pain which he diagnosed as cholecystitis. He noticed, during his examination, some dullness to percussion over the right lung base and X-ray showed an extensive shadow in the right lower lobe. After the cholecystitis had subsided, it was found that the dullness to percussion was still present. Further X-rays and tomograms were taken (Fig. I) and the report, which is typical of the others in this series, was as follows:

> "A well-circumscribed mass occupies most of the lower half of the right hemithorax. It is more or less rounded but slightly lobulated and appears to project forwards and laterally from the posterior chest wall. Elsewhere the lung fields are clear. A variety of lesions could cause this appearance. The most likely would be neurofibroma, but hydatid and various other cysts cannot be excluded."

FIG. I. Lateral radiograph of the chest (Case 5) showing the large circumscribed opacity, posteriorly.

The other four cases presented a similar radiological picture of a well-circumscribed homogeneous opacity, without calcification or fluid levels, but the differential diagnosis

of lesions which could give this picture was so extensive that in reality, radiological examination gave little help in deciding the diagnosis.

Special investigations were carried out to exclude as far as possible hydatid cyst and tuberculoma, but no positive results were obtained. In view of the undecided and possibly serious nature of the lesion, thoracotomy was advised in each case.

portion of the right lower lobe and the other containing a single vein running into the pulmonary ligament and apparently communicating with the systemic circulation.

The tumours, lying so freely in the pleural cavity, were easily removed after ligation and division of the main pedicle and adhesions. The raw area of lung was oversewn and the chest closed with drainage. Convalescence was uneventful.

TABLE I

THIS TABLE GIVES AGE AND SEX OF THE PATIENTS AND THE SITE, SIZE AND NATURE OF ATTACHMENT OF THE TUMOURS TO ADJACENT TISSUES

Case No.	*Age*	*Sex*	*Site*	*Greatest Diameter*	*Remarks*
1	32	F.	Base of lingula	7 cm.	No adhesions — Narrow pedicle
2	38	F.	Anterior edge right upper lobe	10 cm.	Loosely adherent to anterior mediastinum
3	53	M.	Posterior edge right upper lobe	4 cm.	Very small pedicle
4	51	M.	Right lower lobe	14 cm.	Few pleural adhesions
5	55	F.	Right lower lobe	12 cm.	Adhesions to chest wall

OPERATIVE FINDINGS

The operations were performed by Mr. C. J. Officer Brown and Mr. K. N. Morris. The chest was opened by resection of the appropriate rib and in each case a mushroom-shaped tumour was found lying relatively freely in the pleural cavity and attached to the surface of the lung by a short but very vascular pedicle. The site of attachment was variable (Fig. II). Two of the tumours were attached to the right upper lobe, one anteriorly and one to the posterior border, and two were attached to the right lower lobe. The fifth was found arising from the cardiac surface of the base of the lingula of the left lung. In general there was only one thick pedicle with a few small vascular adhesions to the lung but in two cases there were also some denser adhesions connecting the tumour with the chest wall. In one of these, when the adhesions were divided, it was seen that there were two pedicles, one containing many blood vessels and continuous with the apical

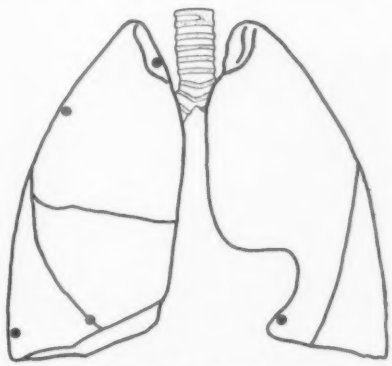

FIG. II. Diagram illustrating the sites of attachment of the tumours.

PATHOLOGICAL FEATURES

Macroscopically, the tumours presented a similar appearance (Figs. III, IV and V). They varied in size from 4 cm. to 14 cm. in their greatest diameters and were roughly pyramidal in shape, with the root of the

pedicle at the apex of the pyramid. The surfaces were slightly convex and separated from each other by sharply defined borders, along some of which were small fringes of papillae. In general, the surfaces were smooth and glistening and were pinkish-white in colour. One of their most prominent features was the presence of numerous large veins coursing over the surface, radiating away from the root of the pedicle where they were largest.

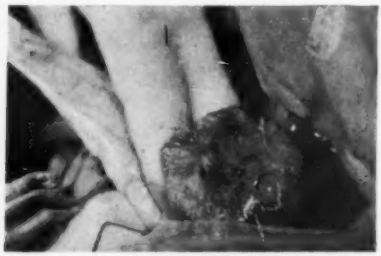

FIG. III. Lingula with attached tumour exposed at operation (Case 1).

When cut across through the root of the pedicle, the tumours were seen to be completely encapsulated by a layer of condensed fibrous tissue with a thin covering of pleura. The main mass of the tumour was composed of white hyaline fibrous tissue with a whorled pattern. In some areas there was evidence of early hyaline degeneration but there was no calcification or cyst formation.

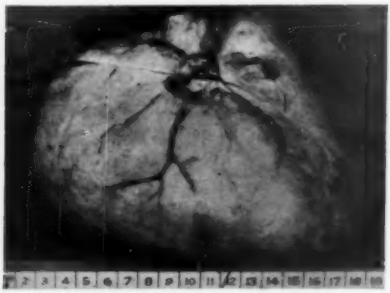

FIG. IV. Photograph of tumour from Case 4.

Microscopically, the tumours consisted of bundles of large, spindle-shaped cells with, in parts, a tendency to whorling (Fig. VI). The stroma was variable, being scanty in the more cellular areas but in the relatively acellular areas was represented by well-developed fibrous tissue. No ganglion cells could be found and special stains did not reveal the presence of any muscle fibres. Histologically, therefore, the tumours were regarded as being composed only of fibrous tissue and, in view of the few mitotic figures and the lack of any other evidence of malignancy, both microscopically and macroscopically, they have been considered as benign fibromas.

FIG. V. Photograph of tumour from Case 5 showing cut surface.

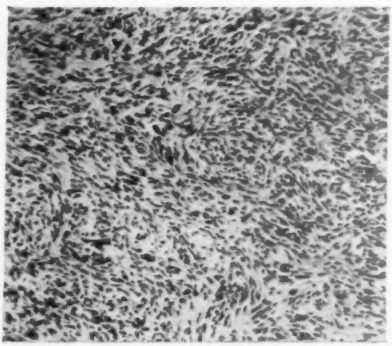

FIG. VI. Photomicrograph of a section from Case 3. (x 90)

DISCUSSION

The remarkable similarity between the tumours just described has prompted the presentation of this series and because of their unusual appearance and situation it is interesting to speculate on their exact nature.

Although, macroscopically, the cut surface bore some resemblance to a fibromyoma this was not confirmed by the microscopic sections which showed the tumours to consist only of fibrous tissue of varying degrees of cellularity. While it is always difficult to distinguish between fibrous tissue and muscle in

haematoxylin and eosin sections, a careful search of the sections, especially of bundles cut in cross section where the centrally placed nuclei and abundant pink cytoplasm of muscle fibres are more easily recognized, failed to reveal any such fibres. This was confirmed by sections stained specifically for muscle cells and, although these stains are occasionally unreliable, they are of some value as confirmatory evidence.

It was also necessary to differentiate the tumours from the neurofibromas, neurilemmomas and ganglioneuromas. Although both macroscopically and microscopically there was some whorling of the fibrous tissue, it was not the tight whorling seen in the neurofibroma, nor was there any palisading of the nuclei so characteristic of the neurilemmoma. Ganglion cells could not be found and the tumours themselves, attached to the borders of the lung, were well removed from peripheral nerves and the sympathetic chain, so that the possibility of their being derived from neural tissue is unlikely.

If then the tumours are accepted as being composed exclusively of fibrous tissue, from what structure do they arise? Their situation suggests three possibilities.

1. They may arise as an ectopic lung bud.
2. They may be the result of inflammation.
3. They may be a true neoplasm.

The first possibility has little to support it except the site, the consistently similar appearance and the fact that, *in situ*, these tumours resemble a mal-developed lobe of the lung. This resemblance, however, is based mainly on their shape, which is probably the result of pressure from the surrounding lung during growth. Histologically, there were no alveoli, cartilage or bronchial epithelium recognized, and while an origin from an ectopic lung bud cannot be definitely excluded, it must be regarded as being most unlikely.

It is also unlikely that an inflammatory process would give rise to such an isolated and well-circumscribed mass of fibrous tissue. At operation, the pleural cavities were quite free from adhesions, and there was no evidence of old inflammation except for the few adhesions intimately associated with the tumours.

Both the histology and the macroscopic picture are compatible however with these tumours being true neoplasms, and they have finally been regarded as benign tumours of fibrous tissue or, for want of a better name, "fibromas"—a term which, nevertheless, gives some indication of their origin and structure.

It is interesting to note that most of the previously reported intrathoracic fibromas have been found densely adherent to, and apparently arising from, the parietal pleura overlying the diaphragm. Price Thomas and Drew, however, have recently reported six cases where the tumours were arising beneath the visceral pleura and were macroscopically similar to those in our own series. But, whereas the patients in our series were relatively symptom free, Price Thomas and Drew found that three of their six cases presented with severe joint pains and swelling of the hands and feet, and showed varying degrees of hypertrophic pulmonary osteo-arthropathy. Others complained of cough, dyspnoea and chest pain, and only one patient was symptomless. It was noticed that the joint changes were completely and dramatically relieved by removal of the tumour in each case.

As only one other report of a case of fibroma of the visceral pleura could be found in the literature, it is not possible to decide what role the tumours themselves play in the production of symptoms, but it would seem that the size of the tumour can play little part as even the largest tumour in our series, measuring 14 cm. across, was completely asymptomatic.

SUMMARY

Five cases of fibroma of the visceral pleura, four of which were detected by mass X-ray survey, are reported.

ACKNOWLEDGEMENTS

I wish to thank Mr. C. J. Officer Brown and Mr. K. N. Morris for their permission to publish these cases and Dr. A. V. Jackson for his help and encouragement during the preparation of this paper.

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CASE OF ADAMANTINOMA OF LOWER JAW

By JOHN MADDERN

Adelaide

THIS case is one of an adamantinoma of the lower jaw in a young woman which was initially treated conservatively but later by resection of the mandible with the insertion of a vitallium plate as a temporary internal prosthesis with good functional and cosmetic results.

FIG. I. Patient before operation.

FIG. II. Patient three months after operation.

CASE HISTORY

In January, 1950, Miss P.C., aged 18, presented with a swelling of the lower jaw in the region of the last molar tooth. It had been present for seven months and was first noticed soon after the extraction of the last molar tooth. At this time X-ray investigation revealed a large cystic area involving the angle of the mandible and extending some distance up the ramus. At operation the cyst was exposed by removing the alveolar margin, the cyst lining removed and the cavity was packed. The cyst wall was examined histologically and showed an epithelium disorganized by acute inflammation and underneath chronic inflammatory changes with fibrosis. The diagnosis of dental cyst was made. Healing of the cavity was, however, delayed. The patient reported back eighteen months later when clinical and radiological examination showed that the cyst had recurred in the body and ramus of the jaw. A biopsy revealed that the epithelium now showed proliferation of a basal cell pattern and the diagnosis of adamantinoma was made. At this time the patient was about to be married and the conservative treatment of removal of the cyst lining and currettage of bone was carried out. The cyst recurred again and was curretted on a further two occasions. In May, 1954, the patient reported back having recently had a child. During the pregnancy the tumour had increased rapidly in size (Figs. I and III). On this occasion resection of the mandible was suggested and agreed to. Immediately preceding the operation the cyst ruptured into the mouth. The operation was conducted through a four-inch incision in the neck. The mandible was exposed and sectioned half an inch anterior to the tumour and the remainder of the body and ramus were excised. To restore the contour of the face a six-inch Sherman plate was bent to conform with the shape of the mandible and was fixed anteriorly by two screws, the free end articulating in the glenoid cavity (Figs. IV and V). The masseter and pterygoid muscles were sutured to the plate. The mucous membrane of the mouth was closed and the wound in the neck closed with drainage. To prevent lateral displacement of the jaw, cap splints which had been fitted preoperatively were wired together. The wires were

replaced with rubber bands after seven days and the splints were removed ten days after this. Primary healing of the mucosa and skin was obtained. Three months later the patient has good facial contour (Fig. II), good range of movement, no lateral deviation of the mandible in talking and eating. She has, however, some degree of facial nerve paralysis affecting the lower lip. This can be seen in Fig. II). It is proposed at a later date to replace the plate with a bone graft.

FIG. III. Radiograph showing extent of cyst immediately pre-operatively.

DISCUSSION

The treatment of adamantinoma by the conservative measures of excision and curettage failed in this case. While this is in accordance with accepted principles, one can understand the reluctance of a surgeon to suggest, and a patient to accept, resection of the jaw in a young woman. However, it is felt that provided some adequate internal splint can be provided, the cosmetic and functional disadvantages are removed. The use of a vitallium prosthesis in a case of adamantinoma is warranted as primary union can be confidently expected, both in skin and mucous membrane. Radiotherapy is not to be used and once adequately removed no recurrence is anticipated. Several vitallium prostheses are described (Conley, 1951) but the use of a Sherman plate bent to the required shape is a simple prosthesis which can be obtained in any surgical centre.

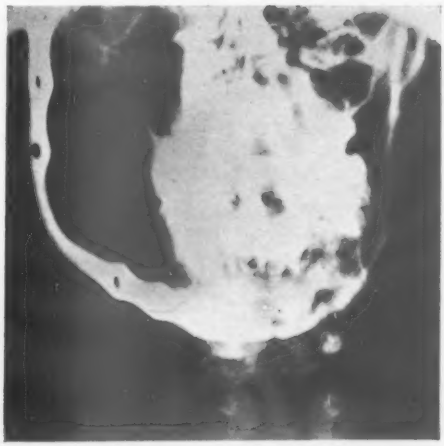

FIG. IV. Anterior radiograph showing position of plate.

FIG. V. Lateral radiographic view of position of plate.

SUMMARY

This is a case report of an adamantinoma treated by resection of the mandible with insertion of a Sherman plate as an internal prosthesis.

ACKNOWLEDGEMENTS

I wish to thank Mr. Alan Lendon for his permission to publish this case and to Miss G. Walsh for assistance in preparation of illustrations.

REFERENCE

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MEGAOESOPHAGUS WITH MEDIASTINITIS TREATED BY PARTIAL OESOPHAGO-GASTRECTOMY

By V. J. KINSELLA

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VARIOUS forms of instrumental dilatation, and, failing these, Heller's operation, will provide sufficient relief for nearly all patients with achalasia and oesophagectasia. But when the dilatation and hypertrophy of the oesophagus have reached extreme dimensions, and when the tortuosity is so great that "food traps" are formed and do not empty when the patient is in the erect position, and when inflammation has spread through the thickened wall of the oesophagus to the mediastinum, simpler methods of treatment may fail and a more radical operation may be required. Such a problem was, as follows:—

On 18th December, 1949, Mr. J.L., aged 48, was admitted to St. Vincent's Hospital, Sydney, complaining of recurring influenza-like attacks for three years, with shivering, joint pains, headaches, and fever 101°F to 102°F, during which he was unable to work. The attacks had been recurring more frequently and the present attack had lasted for one month. The patient had dysphagia due to cardiospasm, which had been recognized twenty years previously, and which had been treated, with little relief, by instrumental dilatation.

FIG. I. Opaque meal before operation.

FIG. II. The resected specimen.

Fluoroscopy and barium swallow showed the enormous tortuous oesophagus, its right border actually touching the chest wall laterally, with two fluid-traps, the lower one resting on the right dome of the diaphragm and emptying only when the patient inclined to the left, and an upper one emptying only when he inclined to the right (Fig. I).

Mr. H. Windsor examined the patient in consultation and, after bronchoscopy and oesophagoscopy, reported intense engorgement of the right bronchial tree, normal bronchograms, and a "gigantic oesophagus filled with most offensive decomposing

food. There were numerous sacculations. The cardio-oesophageal orifice was not visualized."

On 7th February, 1950, Heller's operation (cardiomyotomy) was performed through a midline epigastric incision. The oesophagus presented easily after it was drawn down. The muscular coat of the stomach retracted normally when cut, but the muscular coat of the oesophagus did not retract normally, and, because of inflammatory infiltration it was not easily distinguishable from the mucosa. The incision in the wall of the oesophagus was carried through all coats and the mucosa was then sutured. The patient left hospital twelve days later.

FIG. III. Opaque meal five weeks after operation.

The patient was re-admitted on 11th August, 1950. Since his last operation his dysphagia had improved, and he had gained weight, but he had had two more febrile attacks, and, in the present one, his temperature reached 103°F to 104°F.

Dr. Bruce Hall and Dr. J. Sherwood were of the opinion that the febrile attacks were due to oesophagitis. The patient was treated by penicillin and streptomycin. A mercury weighted tube was swallowed and finally passed into the stomach and was used for feeding. Oesophageal lavage was carried out daily. The patient's general condition improved, and he became afebrile.

On 26th September, left thoracotomy was done, through the bed of the eighth rib. Thirty ounces of clear straw coloured fluid were removed from the left pleural cavity. The lung was lightly adherent at its base, and there was oedema of the extra-pleural tissues, slight in the costal region, but very marked over the mediastinum, with much fibrin on the pleura in this part. At this stage it was decided, after consultation with Mr. H. Windsor, that the acute inflammatory exacerbation had not sufficiently subsided and the chest was closed.

FIG. IV. Opaque meal nearly four years after operation.

A left thoracotomy was performed on 17th October through the previous incision and the oesophagus was freed from its bed. The enormous cavity left in the mediastinum extended through to the chest wall in the right axillary line. The diaphragm was then divided and the stomach mobilized. A partial oesophago-gastrectomy was accomplished by dividing the oesophagus just below the arch of the aorta, and by dividing the stomach transversely just proximal to the incisura in order to remove most of the acid-bearing mucosa and thus prevent acid peptic-oesophagitis. An end-to-end three-layered anastomosis was then made between the pyloric end of the stomach and the remainder of the oesophagus. Convalescence was complicated by haematemesis for which transfusion was required nine days after operation. The patient left hospital on 9th December, 1950.

The specimen removed is shown in Fig. II, after shrinkage by fixation. Our pathologist, Dr. J. Garvan, reported thickening and para-keratosis of the squamous epithelium and pronounced inflammatory changes in the sub-mucosa. "I cannot find any ganglion cells in the dilated portion of the oesophagus or in the block taken at the cardio-oesophageal junction."

On 23rd November, 1950, a skiagram was taken after barium swallow, Fig. III, and it was noted that the suture line had retracted upwards as high as the manubrium sterni.

The patient was re-admitted to hospital on 24th May, 1954, for haemorrhoidectomy.

On 22nd July, 1954, he was shown at a British Medical Association meeting at the hospital. Since his oesophago-gastrectomy he has had no dysphagia and no febrile attacks; he has gained over one stone in weight, and, since January, 1951, has worked continuously at his job as a fitter. During the first twelve months after operation he had attacks of nausea, weakness and palpitations, especially after meals, relieved by lying down. These still come occasionally but are very mild. There is sometimes regurgitation of fluid into the mouth, sometimes bitter, sometimes sour, and this is relieved at night by raising the head of his bed. He describes the result of operation as excellent.

Fig. IV shows a skiagram taken on 15th June, 1954. The junction of stomach and oesophagus can be seen on the level of the manubrium sterni.

SUMMARY

1. A case of megaoesophagus is described.
2. The first point of interest is the large size of the oesophagus, reaching the lateral chest wall.
3. The second point is the influenza-like febrile attacks caused by it.
4. The third point is its treatment by oesophago-gastrectomy, after instrumental dilatation and Heller's operation had failed. Results are reported nearly four years later.

Books Reviewed

DIE GYNAKOLOGISCHEN OPERATIONEN UND IHRE TOPOGRAPHISCH-ANATOMISHEN GRUNDLAGEN.

By HEINRICH MARTIUS. Seventh Edition. Stuttgart, Germany: Georg Thieme Verlag, 1954. 10½" x 5¾", xvi plus 434 pp., 450 illustrations.

This volume is a typical product of German anatomy and surgery. It reminds one of a Spalteholz atlas of anatomy as well as a Kirschner textbook of operative surgery.

Detail of anatomy is described and illustrated as a preliminary to the description of an operation.

The author eschews the mechanical retractor as productive of ischaemia and favouring infection. Hand retractors are advised.

Drainage of the infected pelvis is advised, both abdominal and vaginal. A warning is given against the indiscriminate use of sulphonamide powder intraperitoneally. The illustrations of the Wertheim operation are excellent.

There is much sound advice on the indications for the various operations described.

Standard procedures are pictorially represented, so that it all looks so easy. A little stress on the dangers to bladder and ureters and how to avoid them would be appreciated. However, the many beautiful illustrations and adequate text on the repair of vesico-vaginal and uretero-vaginal fistulae to some extent atone for some omissions. Operations for stress incontinence, as well as the many well-known methods of vaginal plastic repair are beautifully depicted. In fact the book can be regarded as an atlas of operative gynaecology in the older well-known German style.

Simple operations such as appendicectomy, colostomy, and herniotomy are also described. The radical pelvic evisceration is wisely referred to only, it being hardly within the province of the single-handed gynaecologist.

The book can be recommended to the general surgeon as well as to the specialist.

TUMOURS OF LYMPHOID TISSUE.

By GEORGE LUMB, M.D. Edinburgh: E. and S. Livingstone Ltd., 1954. 9¾" x 6¼", viii plus 204 pp., 194 illustrations, 18 tables. Price: 37/6 stg.

This work is a concise and well-documented presentation of some of the diseases of lymphoid tissue. Any contribution which will add to our knowledge or clarify our concepts of these diseases deserves special attention.

There is an introductory section on the structure, function and significance of lymphoid tissue, a short historical survey, a chapter dealing with terminology and classification and a review of the incidence of tumours of lymphoid tissue. Subsequently there are chapters on the individual conditions which are included under this heading of "tumours" and there is a good discussion of "problems of diagnosis" and the effects of therapy on these tissues.

The general presentation of the material is clear and it is apparent that the author has had a considerable experience of these conditions. An important point is that he has kept his classification within the bounds of present-day views, that is to say, he has not introduced, with the exception of "reticular lymphoma," any new terms.

As the name of the book implies he begins with the proposition that the various conditions are tumours. This in itself will present a difficulty to some who have studied these conditions for many years and the opinion that " it is felt that no satisfactory evidence is available in support of any other theory" is very poor grounds for reaching the conclusion that the conditions are tumours. It is naive to infer that those who regard Hodgkin's disease as being some type of inflammatory condition are wrong because they may be compared with some of the older writers whose views have since been shown to be incorrect (p. 20). All that they have in common is that the author disagrees with them.

If, however, one accepts the material in this book and does not allow the author's predilection for an hypothesis of neoplasia, which is as uncertain as any other, to weigh too greatly, then the book is a very valuable one. This applies particularly to post-graduates. For students it would be necessary to indicate the shortcomings inherent in the whole-hearted acceptance of one point of view.

As mentioned previously there is evidence that the author has investigated a large amount of material and the summaries and tables are particularly valuable. The illustrations are clear and well produced and will be found valuable by all students. The text is clear and easy to read and there is an adequate index.

PREFRONTAL LEUCOTOMY AND RELATED OPERATIONS.
Anatomical Aspects of Success and Failure.

By ALFRED MYER, M.D., and ELIZABETH BECK. London: MacMillan and Co. Ltd., 1954. 9¾" x 7½", 60 pp., 20 illustrations (including 8 plates), 18 tables. Price: 10s. 6d. net.

This volume was published by the William Ramsay Henderson Trust which was founded in 1832 for the advancement and diffusion of the Science of Phrenology. Within a few decades, phrenology as a science foundered because it pushed its theories too far in advance of accepted knowledge, and was carried to extreme lengths by unqualified practitioners. Over a century has passed, but the problems associated with localization of function within the brain remain largely unsolved. In spite of this, a new branch of surgery has grown up and many thousands of operations have been performed upon the frontal lobes by surgeons whose knowledge of the function of these structures is still little better than that of the phrenologists a hundred years ago. Many may have felt that they would have been almost as happy with the phrenologists' map of the faculties of the brain as with modern views on localization of functions in this region. For the frontal lobes still baffle the investigators, and there is still no complete answer to Freeman's question whether the frontal lobe functions as a mosaic of

regionally separated faculties, integrated by association tracts or by the mass action of the whole lobe in which the various regions are all of equal potential in this respect.

In spite of the inadequate knowledge of localization of function, the lack of correspondence between surface landmarks and the cyto-architectonic areas of the underlying cortex, together with the difficulties in assessing the extent and location of the various cuts performed at either open or closed operations, much can be gained by a careful statistical survey of clinical and post-mortem material. In this volume the authors have analyzed the anatomical extent of the incision in the white matter of the frontal lobe and the extent of the associated degenerative changes in the thalamic nuclei and elsewhere, which were observed in the brains of 102 patients who have been subjected to leucotomy. These anatomical changes are correlated with the clinical effects of the operation, particularly those aspects directly related to success or failure of the operation.

From the purely anatomical aspect, the investigations have done much to clarify some of the connections of the frontal lobe and have confirmed the interdependence of the frontal region and the thalamus and the hypothalamus and indirectly with the rhinencephalic centres.

In the clinical field, one of the most significant findings related to the amount of white matter divided. It is evident that to produce a satisfactory result, the amount of white matter cut must be adequate, but providing an adequate section is performed, anything in excess of this does little to improve the result. Nor is it always clear which fibres should always be cut. It is generally conceded that the medial, orbital and cingulate portions are most significant, but full social recovery did occur without involvement of these areas.

There was a definite correlation between personality change and clinical improvement which depended upon a regional factor. Marked personality change being linked with section of the lateral and central portions of the white matter at the plane of section of the standard operation and more directly still with involvement of the cortex.

The number of cases studied and the selection of groups gave no opportunity for the assessment of suitability for operation or of assessing subsequent improvement. There were no studies on the effectiveness of the operation in combatting intractable pain. But the work is a valuable contribution to anatomical, surgical and psychiatric knowledge in this field.

TEXTBOOK OF OPERATIVE SURGERY.

By ERIC L. FARQUHARSON, M.D., F.R.C.S.(Edin.), F.R.C.S.(Eng.). Edinburgh: E. and S. Livingstone Ltd., 1954. 9¾" x 6", viii plus 853 pp., 627 illustrations. Price: 75s. net.

The appearance of a new single volume, single author, "Textbook of Operative Surgery" is the more welcome, because it is now so unusual. The author set out to describe "All the operations which the junior in general surgery is likely to undertake himself."

He has included those operations in specialized surgery which sometimes, even in these days, of necessity, fall to the lot of the general surgeon. There is, in addition, a review of the surgical anatomy involved and a discussion on the surgical management, both pre- and post-operative.

In fact, so ample is the scope of the book in general, that it might be better titled "A Textbook of Surgical Treatment."

The book is written with pleasing, uniform lucidity of style, is profusely illustrated and surgically sound.

It most creditably achieves the object which the author sets himself and should be a useful basic surgical textbook for the young general surgeon.

LOCAL ANALGESIA: BRACHIAL PLEXUS.

By R. R. MACINTOSH, M.A., M.D., F.R.C.S., F.F.A.R.C.S., D.A., and WILLIAM W. MUSHIN, M.A., M.B., B.S.(Lond.), M.R.C.S., F.F.A.R.C.S., D.A. Third Edition. London: E. and S. Livingstone Ltd., 1954. 7½" x 5", 62 pp., 32 illustrations, including 15 in colour. Price: 10s. 6d. net.

This is the third edition of this monograph to appear in 10 years, the previous edition appearing under the title "Local Anaesthesia: Brachial Plexus" in 1947.

It has become the standard text on the subject and is now brought up to date by revision of minor anatomical points. In addition references are made to lignocaine (xylocaine) and hyaluronidase (hyalase) which have come into common usage since the publication of the previous edition.

The method of presentation of this essentially practical subject is almost entirely by diagram, and the book can be recommended to any person desiring to acquire skill in blocking the brachial plexus with local analgesic agents.

A SYNOPSIS OF OBSTETRICS AND GYNAECOLOGY.

By ALECK W. BOURNE, M.A., M.B., B.Ch., F.R.C.S. Eleventh Edition. Bristol, England: John Wright and Sons, 1954. 7¼" x 4¾", vii plus 536 pp., 170 illustrations. Price: 25s. net.

This book can serve a useful purpose in that unhappy period of our medical lifetime—those tense ten weeks before the final examination. Anything that is of use at that stage of our career justifies its existence.

Before reading the book one would hope that fuller works had been consulted, and after graduation one would hope for the same thing. In general the advice given in it is sound; improvements could be by deletion rather than by additions.

Decapsulation of the kidneys is mentioned in the treatment of anuria. There is still mention of the neurotic variety of *hyperemesis gravidarum*. Nervous women are affected but their affliction is based on the physical existence of a pregnancy; the attitude that the condition is neurotic can delay treatment.

In these antibiotic days it is possible to clear the urine of all signs of infection after pyelitis of pregnancy, despite what authors say.

Despite these relatively minor differences of viewpoint students will continue with benefit to turn to this book for help in the last sprint before finals.

Book Received

HANDBOOK OF MEDICAL TREATMENT.

Edited by MILTON J. CHATTON, A.B., M.D., SHELDON MARGEN, M.A., M.D., HENRY D. BRAINERD, A.D., M.D., and sundry authors. Fourth Edition. California, U.S.A.: Lange Medical Publishers, 1954. 7" x 4", 569 pp. Price: $3.00.

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